

MOOD CONSTRAINT ON SELF-APPRAISAL: TOWARD BRAIN-BASED
ASSESSMENT OF DYSFUNCTIONAL THINKING

by

ALLISON C. WATERS

A DISSERTATION

Presented to the Department of Psychology
and the Graduate School of the University of Oregon
in partial fulfillment of the requirements
for the degree of
Doctor of Philosophy

June 2015

DISSERTATION APPROVAL PAGE

Student: Allison C. Waters

Title: Mood Constraint on Self-Appraisal: Toward Brain-Based Assessment of
Dysfunctional Thinking

This dissertation has been accepted and approved in partial fulfillment of the
requirements for the Doctor of Philosophy degree in the Department of Psychology by:

Don M. Tucker	Chairperson
Jennifer H. Pfeifer	Core Member
Maureen Zalewski Renier	Core Member
Mark Johnson	Institutional Representative

and

Kimberly Andrews Espy	Vice President for Research and Innovation; Dean of the Graduate School
-----------------------	--

Original approval signatures are on file with the University of Oregon Graduate School.

Degree awarded June 2015

© 2015 Allison C. Waters

DISSERTATION ABSTRACT

Allison C. Waters

Doctor of Philosophy

Department of Psychology

June 2015

Title: Mood Constraint on Self-Appraisal: Toward Brain-Based Assessment of
Dysfunctional Thinking

The goal of the current research is to characterize the neural mechanisms of mood-cognition interaction in self-evaluative decision-making. Self-evaluation is mood state dependent. A transient decrease in positive self-evaluation bias may co-occur with sad mood. In clinical depression this decrease is lasting and exaggerated. The act of self-evaluation engages frontal lobe mechanisms of emotion regulation, but it remains unclear how these constraints on cognition become pathological in depression. In four studies, dense array electroencephalography (256 dEEG) was recorded as participants performed a self-appraisal task. Analysis of the event-related potential was closely aligned with psychometric methodology. Findings elaborate on network models of neural self-regulation and depression pathology. Characterization of frontal lobe mechanisms in this context provides insight into the neural basis of adaptive and dysfunctional social behavior.

CURRICULUM VITAE

NAME OF AUTHOR: Allison C. Waters

GRADUATE AND UNDERGRADUATE SCHOOLS ATTENDED:

University of Oregon, Eugene, Oregon
Stanford University, Palo Alto, California

DEGREES AWARDED:

Doctor of Philosophy, Clinical Psychology, 2015, University of Oregon
Master of Science, Psychology, 2009, University of Oregon
Bachelor of Science, Biological Sciences, 2007, Stanford University

GRANTS, AWARDS, AND HONORS:

Ruth L. Kirschstein National Research Service Award, National Institutes of
Mental Health, F31MH094052, 2012-2014

Hendricks Scholarship, University of Oregon, 2011

University of Oregon General Scholarship, 2009-2011

Vice Provost for Undergraduate Education Grant, Stanford University, 2006

BioX Undergraduate Research Grant, Stanford University, 2005

Parsons Foundation Internship, University of California, Berkeley, 2003

PUBLICATIONS:

Waters, A.C. & Tucker, D.M. (2013) Positive and negative affect in adolescent self-evaluation: Psychometric information in single trials used to generate dimension-specific ERPs and neural source models. *Psychophysiology*, 50, 538-549.

Waters, A.C. & Tucker, D.M. (2013). Self-Regulation of Neural Development. In F. Grouzet, U. Muller & B. Sokol (Eds.), *Self-Regulation and Autonomy: Social and Developmental Dimensions of Human Conduct*. New York: Cambridge University Press

Waters, A.C., Song, J., Luu, P., & Tucker, D.M. (2012) The importance of single trials; Temporal and spatial resolution in event-related research. *Developmental Neuropsychology*, 37(6), 545-558.

- Waters, A.C. & Tucker, D. M. (2012). Neurophysiology of Motivated Learning. In *Encyclopedia of the Science of Learning* (Part 14, pp. 2455-2457). New York, NY: Springer
- Barretto, R.P.J., Ko, T.H., Jung, J.C., Wang, T.J., Capps, G., Waters, A.C., Ziv, Y., Attardo, A., Recht, L., & Schnitzer, M.J. (2011). Time-lapse imaging of disease progression in deep brain areas using fluorescence microendoscopy. *Nature Medicine*, 17, 223-228.
- Tucker, D.M., Waters, A.C., Holmes, M.D. (2009). Transition from cortical slow oscillations of sleep to spike-wave seizures. *Clinical Neurophysiology*, 120, 2055-2062.

ACKNOWLEDGMENTS

I wish to express sincere appreciation to Dr. Tucker for his mentorship. I also thank the members of my dissertation committee for their valuable feedback on this manuscript. This research was supported by the National Institutes Of Mental Health, grant # F31MH094052.

TABLE OF CONTENTS

Chapter	Page
I. INTRODUCTION	1
Chapter Outline.....	3
II. THEORETICAL BACKGROUND	5
Part I: Affective Cognition.....	6
Part II: A Model of Affective Self-Regulation in the Frontal Lobe	11
Part III: Toward Brain-Based Metrics of Neural Self-Regulation.....	17
III. PSYCHOMETRICS IN EEG RESEARCH	22
Part I: A Neuropsychometric Approach	23
Part II: Principal Components Analysis in Event-Related Potential Research.....	29
Part III: Introduction to Study 1, 2 and 3	35
IV. SELF-EVALUATION IN DEPRESSION	41
Methods and Materials.....	42
Statistical Decomposition	43
Statistical Analyses	44
Results.....	45
Summary of Results.....	52
V. SELF-EVALUATION AND OTHER-EVALUATION.....	56
Methods and Materials.....	57
Statistical Analyses	59
Results.....	61
Summary of Results.....	68

Chapter	Page
VI. SELF-EVALUATION AND SEMANTIC CATEGORIZATION	74
Methods and Materials.....	75
Statistical Analyses	77
Results.....	78
Summary of Results.....	85
VII. SUMMARY AND SYNTHESIS	87
Control-Systems Framework for Interpreting Results.....	89
Frontal Lobe Mechanics Instructing Treatment for Depression.....	90
Investigating the Neural Mechanisms of Affective Cognition	92
Factor Analysis in ERP Research	93
Depression Neuropathology in Self-Evaluative Cognition.....	94
Affective Dimensionality and Self-Evaluative Behavior	95
Neural Correlates of Self-Regulation in RVL PFC	96
Tentative Interpretation of the Component Structure	98
Conclusions.....	103
REFERENCES CITED.....	105

LIST OF FIGURES

Figure	Page
1. Schematic of neuropsychometric approach to ERP analysis	25
2. Topographic maps of PA- and NA-weighted ERPs and statistical difference	27
3. Source waveforms in selected Brodmann areas (BAs).....	28
4. Stimulus presentation in Study 1	43
5. Correlation of % negative self evaluation and scores on the Beck Depression Inventory (BDI).	45
6. ERP signature of self-evaluative cognition.....	48
7. Decomposition of the posterior positivity in Study 1	50
8. Topographic maps of grand average amplitude for three components.....	51
9. Right ventral lateral prefrontal cortex (RVLPFC) activity and symptom severity	53
10. Schematic of stimulus presentation in Study 2.....	58
11. ERP signature of evaluative cognition in Study 2	62
12. Decomposition of the posterior positivity in Study 2	65
13. Effects of reference and valence in component amplitude.	66
14. Correlation of PA and amplitude over ventral lateral prefrontal cortex	69
15. Schematic of stimulus presentation in Study 3	76
16. Channels selected for the correlation analysis	78
17. ERP signature of evaluative cognition in Study 3	80
18. Decomposition of the posterior positivity in Study 3	82
19. Effects of task type and valence in component amplitude.....	83

Figure	Page
20. Replication of factor structure across three studies	94
21. Topography of component amplitude across studies.....	98

LIST OF TABLES

Table	Page
1. PANAS word items and loading scores on PA and NA	24
2. Demographic and clinical characteristics in Study 1	46
3. Correlation of trait affect and self-evaluative behavior in Study 1	47
4. Correlation of trait affect and evaluative behavior in Study 2	61
5. Correlation of trait affect and amplitude over ventral lateral prefrontal cortex.....	68
6. Correlation of trait affect and self-evaluative behavior in Study 3	79

CHAPTER I

INTRODUCTION

In a 2008 report, the World Health Organization identified depression as a primary contributor to the global burden of disease (World Health Organization, 2008). Depression accounts for the greatest number of years lost to disability worldwide. In the US, this amounts to \$36 billion dollars in lost wages, every year (Kessler, Akiskal, & al., 2006). It is estimated that 20% of depression sufferers who seek help do not respond to treatment, and up to 60% do not achieve complete remission (Kessler, Burglund, & al, 2003). Despite the human and economic cost of this psychiatric illness, we do not currently understand depression well enough to treat it effectively.

Research in medicine and neuroscience has provided evidence of depression pathology in the brain. Findings do not easily coalesce, however. It appears that depression is a heterogeneous disorder, both in symptom profile and neural pathology (Fitzgerald, Laird, Maller, & Daskalakis, 2008). A current challenge in clinical neuroscience is to find a parsimonious explanation for seemingly disparate results. In depression research, it has become necessary to model neural dysfunction at the level of whole-brain dynamics (Drevets, Price, & Furey, 2008; Mayberg, 2003; Northoff, Wiebking, Feinberg, & Panksepp, 2011; Tucker & Luu, 2007). Depression is thus viewed as a disorder of neural self-regulation, and the complexity therein begins to account for heterogeneity in the symptom profile. This shift in conceptualization, however, requires new approaches to the analysis of neural data. It also suggests new goals for the assessment of neuropathology in clinical practice.

In the current research, I explore novel analytic methods in the study of self-evaluative cognition. Self-evaluation is mood state dependent; positive self-appraisal declines with depression and improves with remission (Rimes & Watkins, 2005). Research in this area has been productive in elaborating on network models of brain function and depression pathology. However, this work has been dominated by metabolic measures of neural activity (Lemongne et al., 2010; Sarsam, Parkes, Roberts, Reid, & Kinderman, 2013; Yoshimura et al., 2013). Electrophysiological measures (i.e., EEG) have been less frequently applied (Poulsen, Luu, Crane, Quiring, & Tucker, 2009), despite offering temporal resolution on the scale of cognitive events. The current strategy

closely aligns EEG analysis with psychometric methodology. Psychometric research has made important contributions to basic psychological science, as well as to the assessment of psychological phenomena (e.g., Watson & Tellegen, 1985). Though beyond the scope of the current investigation, this combination of psychometric and EEG methodology is directed toward future development of brain-based assessment tools.

The goal of the current research is to characterize the neural mechanisms of mood-cognition interaction in self-evaluative decision-making. In four studies, EEG was recorded as participants engaged in a psychometric self-evaluation task. This task required participants to read adjectives and rate the extent to which the word was self-descriptive. EEG data was then explored with a novel *neuropsychometric* approach and with factor analysis. Both of these analytic strategies were aimed at elucidating network properties of the EEG signature and identifying neural correlates of positive self-appraisal bias. I also investigated mood bias in self-evaluation with experimental contrasts. Results elaborate on a network model of neural self-regulation that appears dysfunctional in depression.

Cogent models of brain pathology are beginning to have considerable influence on the nosology of psychopathological experience. The current diagnostic system is limited by heterogeneity within diagnostic categories and comorbidity across categories. A recent prevalence survey found that just under 50% of depression diagnoses are accompanied by an additional comorbidity (Kessler, Chiu, Demier, & Walters, 2005). A goal of the current investigation is to identify latent, cross-diagnostic factors. In addition to redefining diagnostic boundaries, findings in this domain will impact methods in clinical research (e.g., inclusion criteria) and may suggest alternative treatment or prevention strategies.

The study of maladaptive mood constraint on cognition may also clarify adaptive mechanisms of emotion regulation. Sadness is an appropriate response to failure or loss, and it may guide adaptive coping through reappraisal of self-schema (Lewinsohn, Solomon, Seeley, & Zeiss, 2000). Mood-congruent self-reflection may help people to adapt their expectations and behavior to new circumstances. In depression, however, negative cognitive bias is lasting, debilitating, and resistant to recovery (Rimes & Watkins, 2005). Understanding how this mood-cognition interaction becomes

pathological in depression may contribute to a basic understanding of affective self-regulation.

Chapter Outline

In Chapter II, I provide a theoretical framework for the current research. The is divided into three parts. In Part 1, I introduce a two-dimensional model of emotion: positive affect (PA) and negative affect (NA). I operationalize mood using these constructs for several reasons. A dimensional model of mood provides a means to quantify variation in emotional experience as co-ordinates on a two-dimensional axis. Liability on the PA scale has been associated with mood disorders; low PA is a specific predictor of depression. The affective-arousal represented by PA and NA is fundamental to a control-systems model of brain function. In Part 2, I describe this model of neural self-regulation, which will be used as a framework for interpreting the results of the current research. I conclude Chapter II with a brief discussion about *neurometrics*, brain-based measures of psychological functioning.

The methods developed for the current investigation convolve psychometric analysis with EEG research. In Chapter III, I provide background in psychometric and event-related potential (ERP) methods, and introduce the research strategy. In Part 1, I review a *neuropsychometric* approach to the study of self-evaluative cognition. In this research, the psychometric properties of word stimuli are used to identify neural activity associated with PA and NA. Results suggest network activity in the ERP signature of self-evaluation. In Part 2, I propose an extension of this research: decomposition of the ERP using principal components analysis (PCA). Such factor analytic techniques are commonly used in psychometric research to characterize the latent structure of a construct, such as PA and NA dimensionality in emotion. I propose that the latent structure of the self-evaluative ERP will likewise reflect meaningful dimensions, and that dimensions of neural activity will reflect a control-systems model of brain function.

I conclude Chapter III by describing an empirical approach to this proposal. I derive hypotheses for this research from the ERP literature, wherein ERP components are typically studied in isolation. To avoid redundancy in subsequent chapters, Part 3 also serves as an introduction to three studies. With Study 1, I investigate self-evaluative cognition in depression. Results, provided in Chapter IV, demonstrate that as depressive

symptoms become more severe, positive self-evaluation bias decreases. I identify three latent factors, or components, that may be relevant to this pathological mood-cognition interaction. One component appears exaggerated in the clinical sample, while another is attenuated. I speculate that these results might reflect an exchange in dominance between affect-biased, neural control systems. These findings may provide insight into the neural mechanics of co-morbid symptoms of anxiety and depression.

In Chapter V, I present the results of Study 2, which further characterize mood-cognition interaction in components of the evaluative ERP. Study 2 provides a “neurotypical” baseline for results of Study 1. To better identify aspects of the neural signature that are specific to self-processing, I also introduce a control task. Participants evaluate themselves in one condition, and a public figure, President Obama, in another condition. Individual differences in PA predict self-evaluative behavior, but not evaluation of the President. PA is further implicated as a neural correlate of evaluative cognition. Importantly, the component structure observed in Study 1 is replicated in both Study 2 and Study 3.

The third study in the series is intended for replication of the exploratory findings in Study 2. In Chapter VI, I provide results of this final study in the series. In Study 3, I also introduce a second control condition. This task involves categorizing word stimuli; participants must engage in reading and decision-making, but the extent of social evaluation is decreased. Effects of this contrast appear in anterior channels implicating the frontal lobe in self-specific processing.

In Chapter VII, I review and synthesize results from the neuropsychometric analysis, as well as Studies 1-3. Findings are interpreted within a control-systems framework of neural self-regulation. The dimensional structure of the decision-making ERP suggests three consecutive stages of processing that involve mood-cognition interaction. These findings may differentiate aspects of neural self-regulation in self-evaluation that are exaggerated in anxiety, and attenuated in depression.

CHAPTER II

THEORETICAL BACKGROUND

In this chapter, I present a theoretical framework for the current research. To begin, I provide rationale for a dimensional approach to the study of emotion. I introduce a two-dimensional model of affective-arousal; positive affect (PA) and negative affect (NA). These constructs are used to describe individual differences in trait mood. The PA scale is a continuum of arousal with valence. It spans from depression to elation. The NA continuum spans from anxious to calm. I also discuss the convergence of PA and NA with other measures of affective experience, such as personality, behavior and the perception of emotion. In clinical populations, low trait PA predicts symptoms of mood disorders, while NA is aligned with anxiety disorders and general distress. A goal of the current research is to test the predictive specificity of these mood dimensions in healthy and mood disordered self-evaluative behavior.

In the second half of this chapter, I present a model of brain function wherein dimensions of affective-arousal, PA and NA, are fundamental to self-regulatory dynamics. I propose that self-regulation in the brain involves competing neural systems. These are engaged in a dynamic of reciprocal motivation and constraint, which is fundamentally affective. I provide a brief overview of this control-systems model of neural self-regulation, beginning with the visceral and somatic divisions of the brain. The frontal lobe can be further divided into a dorsal cortico-limbic system, and a ventral cortico-limbic systems. The limbic core of the dorsal system provides a PA bias that drives projective, impulsive functions. Ventral functions are more responsive to the environment, and ventral limbic structures drive this vigilance with an NA bias. Though basic in its representation of frontal lobe functioning, I will use this control-systems model as a theoretical framework for the current research.

To illustrate the application of this framework, I then provide an example of dorsal and ventral reciprocity in motor action regulation. I extend these properties of motor control in the frontal lobe to psychological phenomena. I describe neurological disorders in which disruption of frontal lobe systems leads to symptoms of depression. I then apply the control-systems model to cognition and consider how affective-arousal biases in the brain provide motivation and constraint on self-evaluative decisions. A goal

of the current research is to understand how dysfunction in neural self-regulation contributes to the symptomatic decline of positive self-appraisal in depression.

I begin the final section of this chapter by considering functional specialization in the left and right hemispheres as it relates to a neural control dynamic. I review evidence of functional and affective specialization in the cortical hemispheres that suggest hypotheses for the current research. I then introduce a measure of frontal lobe activity, alpha asymmetry, that captures trait-like characteristics of emotion regulation. I consider an explanation for this phenomenon within a control-systems framework. To conclude this chapter, I briefly discuss issues in the development of brain-based assessment tools, or *neurometrics*, and contextualize the current research relative to that goal.

Part I: Affective Cognition

Concepts of affect. For something that is so essential to the human experience, emotion is surprisingly difficult to define and measure. In contemporary emotion research, definitions fall roughly into two categories: categorical and dimensional. The current investigation assumes a dimensional model of affect, and tests some related assumptions. Discrete and dimensional theories share a common vernacular, however, so before providing rationale for these assumptions it may be necessary to define some terms: emotion, primary-process emotion, and mood. These words could be synonyms but they are also used to refer to different concepts. According to a dimensional view, however, these concepts coalesce as multiple facets of our core affective nature.

The dominant paradigm in emotion research divides emotions into discrete categories of experience. According to Ekman and colleagues, basic emotions include: happiness, sadness, surprise, fear, anger and disgust (Ekman & Friesen, 1971). The theory posits that these basic emotions are universal, while other emotions are a product of socialization (Izard, 2007). In this context, an *emotion* is characterized as a set of physiological and cognitive criteria that appear in response to a stimulus (internal or external). Emotions have valence, distinct from neutral states of arousal. The onset of an emotion is rapid, but the experience may persist for seconds or minutes (A. Damasio, 2012). Recent research has aimed to associate discrete emotions with a particular set of neural structures, with limited success (Barrett & Wager, 2006; Berridge & Scherer, 2003; Phan, Wager, Taylor, & Liberzon, 2002). A strength of this categorical approach is

its face validity; it fits with folk wisdom and is intuitive. This resulted in many decades of productive investigation, and some of the first forays into the affective brain.

As the field of clinical and affective neuroscience progresses, however, the foundational evidence for basic emotions is being systematically refuted (Russell, 2009; Zachar & Ellis, 2012). One weakness of the categorical approach, germane to the current research, is that it fails to account for overlapping characteristics of mood and anxiety disorders. Pathologies of mood and anxiety are frequently co-morbid, which suggests shared dimensions that cannot be explained with a categorical nosology (T. A. Brown, Chorpita, & Barlow, 1998; Kessler, Berglund, et al., 2005; Lamers et al., 2011). In this sense, the current diagnostic system of psychiatric disorders is aligned with discrete theories of emotion. Although it may seem obvious to the experiencer that these categories exist in nature, it has been difficult to translate this subjective ontology to brain function (Russell, 2009).

This is particularly true for cortical mapping of discrete emotions. In humans, these maps invariably involve functions of the frontal cortex, which evolved according to the selective constraints of a highly social niche (Dunbar & Shultz, 2007). The frontal cortex was also the latest to develop in mammalian cortical evolution. To localize emotion in the frontal cortex, it follows, affective experience is denied to mammals of lower phylogeny. The promising work of Jaak Panksepp, addresses this criticism carefully. Panksepp (2012) posits a theoretical model of emotion systems, derived largely from animal research. Insofar as these systems pertain to specific classes of behavior (e.g., seeking, play), Panksepp's model has been aligned with discrete emotion theories (Zachar & Ellis, 2012). However, his work describes *primary-process emotions* that are phylogenetically conserved and largely subcortical. This subcortical root of affective experience is shown to be a foundation of brain function, which is then elaborated in higher cortical structures (Panksepp & Biven, 2012).

Affective experience involves dynamic movement along a continuous axis (Osgood, 1952; Russell, 2009; Tellegen, Watson, & Clark, 1999). From primary process emotion to personality, individual differences in affect-biased brain function aggregates in trait-like characteristics (Waters & Tucker, 2013a). This aggregate can be recognized as *mood*, an affective bias that is relatively stable for hours or days. In individuals, a

mood tendency, or trait mood, is stable over the lifespan. There is only an abstract transition from trait mood to personality, which can also be measured within the same dimensional framework (Saucier et al., 2014). In the current research, these dimensions of affect are viewed as fundamental properties of the brain. The brain self-regulates through a dynamic balance of reciprocal control systems (Tucker & Luu, 2012). Within this dynamic system, affect provides motivation and constraint on brain processes that give rise to cognition and complex human behavior.

A dimensional model of affect. A dimensional approach to understanding emotion has been guiding psychological research (Barrett & Wager, 2006; Russell, 2009; Sutton & Davidson, 1997; Tellegen et al., 1999; Tucker & Luu, 2007). The current research adopts the terminology of Watson and Tellegen (1985), who pioneered a linguistic approach to the study of emotion.

We can gather important information about affective experience by looking closely at how it is described in words. A linguistic approach to the study of emotion and personality first identifies all descriptor words in a lexicon, by sorting a dictionary, for example. A survey of all words is constructed; a person might be asked to rate the extent to which a word is self-descriptive or descriptive of another person. When a sufficient number of these surveys have been completed, researchers use factor analysis to look for consistent patterns. Responses to some words will be highly correlated (e.g., enthusiastic and proud), and others will not (e.g., determined and afraid). Overall, these correlation patterns show how some words cluster together. The cluster, or latent factor, can be described as the central tendency of the word group. In emotion research, the factor structure describes something fundamental about the dimensional nature of emotional experience.

In psychometric research, there will always be some debate about how many factors to retain in a factor analysis, and how to describe those factors (Fabrigar, Wegener, MacCallum, & Strahan, 1999). Some of these decisions are made statistically, but ultimately, the strength of a dimensional model is determined by its convergent validity and utility as a predictive metric. For example, emotional experience can be described using two factors: arousal and pleasantness (Russell, 1979). This two-factor structure accounts for over two thirds of the variance in large surveys. That is to say, all

emotion words can be characterized with coordinates within this axis. Though conceptually useful and statistically sound, it is difficult to relate this model to biological systems. In a living organism, pleasantness cannot exist when arousal approaches zero.

Alternatively, the arousal and pleasantness axis can be rotated 45 degrees. In doing, the model retains statistical strength but also suggests a new theoretical model based on affective arousal. In this model, the axis are labeled as Positive Affect (PA) and Negative Affect (NA) (Watson & Tellegen, 1985). PA and NA, it follows, are not opposite poles of a bipolar scale. PA represents a continuum from unpleasant, low arousal of depressive nature to pleasant, high arousal of an elated nature. Sadness and lethargy are characteristics of a low PA state, while high PA descriptors reflect pleasurable engagement in the world, particularly the social world. NA, in contrast, reflects a continuum from calmness to anxiety. NA can be described as a measure of subjective distress.

Convergence with PA and NA. Importantly, this affective-arousal model converges with psychometric research in other domains of affective experience. A contemporary of Auke Tellegen and David Watson, Robert Thayer, arrived at a convergent structure though his investigation focused on features of arousal instead of emotion. Thayer (1978) described the continua as tired to vigorous and calm to tense. When focus is on behavior and post-goal seeking emotion is removed, a two-factor model can be described as approach and avoidance orientation (Carver, 2004; Davidson & Irwin, 1999; Gable & Harmon-Jones, 2010). Related constructs also emerge when the investigation is more strictly limited to descriptors of emotion (Watson, Clark, & Tellegen, 1988), or more broadly to personality traits, extraversion and neuroticism (Tellegen, 1988). Although it has also been useful to understand personality relative to five factors (i.e., Big Five), contemporary research indicates that a two-factor structure of personality, described as social propriety and dynamism, generalizes across cultures (Saucier et al., 2014). The PA and NA structure also emerges even when stimuli are not linguistic, such as facial expressions (Stone, 1988). Each of these models contribute important nuance to theoretical research. The extent of convergence, however, suggests something fundamental about affective experience.

The research above derived insight from the way in which people describe their own affective experience or that of an observed other. An important extension of this work is to the appraisal of emotional stimuli, in general. For example, people can assign affective characteristics to pictures, objects or scenes. This literature is quite large. For research purposes, databases of stimuli have been developed so that the psychometric properties of each picture can be broadly assessed and understood. When emotional pictures are arranged on an arousal and valence axis, the distribution typically takes on a circumflex shape; items tend to fall along the diagonals.

The implication is that the PA/NA structure also describes our experience of emotional stimuli. This shouldn't be surprising. We would expect our experience of objects to utilize the same affective mechanisms that we use when we experience our self. Yet in basic research, particularly in cognitive neuroscience, stimuli are contrasted according to valence (i.e., good/bad) and balanced for arousal features. We seek to understand, for example, differences in the neural response to good and bad stimuli. It may be more biologically relevant, however, to instead to define the emotional attribution of experimental stimuli along the dimensions of PA and NA.

Low positive affect is a central characteristic of depression. The goal of this initial psychometric research was deeply theoretical; aimed at understanding the nature of emotional experience. In practice, PA and NA constructs have been validated as a predictor of future wellbeing. The Positive and Negative Affect Schedule (PANAS) is a psychometric designed to score an individual on PA and NA scales (Watson et al., 1988). On the PANAS, PA and NA are each represented by ten words that correlate highly with the respective factor. The metric has undergone rigorous reliability and validity testing, and has been normed in healthy, clinical and developmental populations (Crawford & Henry, 2004; Thompson, 2007).

PA and NA scores predict patterns of self-appraisal with surprising specificity. Poulsen et al., (2009) found PA scores to strongly predict self-appraisal of socially desirable trait words. NA scores, in contrast, better predicted responses to undesirable traits. Scores on the NA subscale also converge with measures of subjective stress-level, frequency of life-stressors, dysfunctional coping strategies and health concerns. PA scores correlate with social success, as well as levels of social activity (Lyubomirsky,

King, & Diener, 2005). Experimental studies suggest a causal relationship between trait positive affect and a number of success-related variables: relationship satisfaction, health/mortality, employment. Low PA scores uniquely predict the likelihood of future depressive episodes (Gencoz & Tulin, 2002). Low trait PA is a specific risk factor for depression.

In clinical populations, PA and NA are differentiated in important ways (Clark & Watson, 1991). NA has been described as a measure of subjective distress and can thus indicate the severity of psychological problems (D. Watson et al., 1995). The NA construct is best aligned with anxiety disorders, although high NA is associated with anxiety, depression and other psychiatric disorders. In very severe depression, however, NA is less predictive, suggesting an important shift in the course of the disorder. Low PA scores discriminate depression from other psychiatric disorders (Clark & Watson, 1991). Depression can be characterized as a disorder of positive affect. Very high PA is associated with manic states, and lability along the PA axis describes bipolar disorder (Lovejoy & Steuerwald, 1995). This unique pathology suggests some biological basis of positive affect that is discrete enough to give rise to bipolar pathology (Gruber, Johnson, Oveis, & Keltner, 2008).

Part II: A Model of Affective Self-Regulation in the Frontal Lobe

The brain regulates itself. If positive and negative affective-arousal dimensions are fundamental to self-reported emotional experience, this dimensionality might also be apparent in the neural mechanisms of emotional experience (Tucker & Williamson, 1984). A goal of the current research is to identify neural correlates of mood-cognition interaction during self-evaluative cognition. Current methods are aimed at characterizing a network model of cortical functioning. In this section, I will outline a basic model of frontal lobe function, wherein dimensions of affective-arousal (positive and negative) are fundamental to self-regulatory dynamics. Though overly simplified, this model provides a framework for speculation on how psychological phenomena, such as self and personality, might emerge from frontal lobe functioning.

A contemporary student of the brain is afforded certain luxuries. While a comprehensive understanding of brain function remains elusive, the field has arrived at some clarity on previously vexing issues. For example, it is clear now that the brain

functions without a conductor; it is a self-regulating system. The brain functions as a dynamic balance of inter-related systems. Each control system provides a force of opposition that sums to an outcome: physiological, behavioral, and psychological.

Though over-simplified, neural dynamics can be described using principals of early ship navigation, or *cybernetics* (Tucker & Luu, 2007, 2012; Waters & Tucker, 2013a). Using cybernetic action, a ship projects a direct course to its destination, but must constantly adapt its planned trajectory to account for environmental conditions. The sum of those opposing forces (i.e., projection and correction) determines the action of the ship, at any given moment. Turning the metaphor back to the brain, the forces in opposition are recognized as fundamentally affective. This assumption is also a luxury of contemporary neuroscience. Once a niche area of study, affect is now viewed as fundamental to brain function. From the most ancient formations of the brain stem and midbrain, to the phylogenetically new cortical sheet, the cybernetic forces of arousal (i.e., motivation and constraint) are inherently affective.

The visceral and somatic brain; inward and outward orientation. Though mood is embodied at all levels of the neural axis, we address this question at the level of the frontal lobe; the area most unique to humans (Berridge & Scherer, 2003). The mechanics of self-regulation in the frontal lobe introduce a third assumption of contemporary neuroscience, still underappreciated: cognitive processes of the highest order emerge from the sensory and motor systems (Tucker, 2007). The cybernetics of motor action also applies to cognition. This observation leads us to develop hypotheses about how the frontal cortex acts to self-regulate not just motor action but also the seemingly ineffable phenomena of the mind.

The brain can be roughly divided into two anatomical-functional divisions. The visceral brain is concerned with sensing internal needs and driving automatic bodily functions. The somatic brain interfaces with the environment. This includes the sensory and motor systems of the posterior and anterior cortex, respectively. There is a cybernetic balance between these two components of the brain, a constant negotiation of internal drives and conflicting environmental demands. The concerns of the outer cortex are largely somatic (e.g., primary sensory and motor cortices, association cortices), although visceral functions are also represented in the innermost cortical areas (e.g., cingulate

cortex, ventral pre-frontal cortex). These innermost cortices, together with subcortical structures of the visceral brain, comprise the limbic system. The visceral-somatic dichotomy spans all levels of the neural axis. With the goal of sketching an anatomical-functional model of the human frontal lobe, however, it will suffice to assign affective drives to the limbic system (e.g., cingulate cortex, amygdala), and somatic concerns to non-limbic cortex (e.g., dorsal prefrontal cortex).

PA and NA bias in dorsal and ventral cortico-limbic systems. The simplicity of this visceral-somatic dichotomy breaks down in areas of the brain where somatic and visceral concerns negotiate. This intersection is well represented in the frontal lobe. In humans, prefrontal cortex is the most recent product of *encephalization*, a process by which evolution elaborates on the anatomy and function of more primitive structures (Glenn Northcutt & Kaas, 1995). Integrated circuitry in the prefrontal cortex is involved in much of what we identify as uniquely human: the ability to inhibit impulses in service of long term goals, complex problem-solving, theory of mind and other forms of social cognition (Nauta, 1993). The frontal lobe, however, is most essentially related to motor action (Goldman-Rakic, Bates, & Chafee, 1992). It follows that the basic mechanisms of motor control provide guiding principals, if not the actual substrate, for higher order psychological processes.

To begin to make sense of this complex structure, it is useful to further subdivide the frontal cortex into subordinate dorsal and ventral divisions. These are the cybernetic moieties of the frontal lobe. It is also at this level of complexity that mood dimensionality in brain function is apparent; processes are biased by PA in the dorsal stream and, by NA in the ventral stream. In the cybernetic self-regulation of the frontal cortex, PA and NA provide the impetus and constraint, respectively. In a recent chapter, Waters & Tucker (2013a) reviewed evidence that differentiates dorsal and ventral aspects of the frontal cortex in form and function. The origin of this cybernetic structure is apparent in theories of cortical evolution (Sanides, 1970), as well as patterns of neural development observed in the embryonic brain (Rakic, 2009). The specialized functions of dorsal and ventral moieties in the mature brain emerge from the particular combination of specialized cell types, neurotransmitter systems, tissue organization and connectivity patterns (reviewed in Tucker & Luu, 2012; Waters & Tucker, 2013a).

The limbic core of the ventral system includes the amygdala, with bidirectional connections to orbital frontal cortex, and ventral cingulate cortex (Barbas & Pandya, 1989; Pandya & Barnes, 1987). The ventral stream derives its negative affective bias from these structures, which motivate vigilant, attentive functions of the somatic cortex. The ventral cortex transitions from visceral to somatic in regions of the frontal pole, lateral cortex and ventral motor areas. This cortical component asserts control over limbic drives, disinhibiting or suppressing negative affect according to environmental demands.

In the dorsal moiety, there is also reciprocal control between somatic and limbic components (Barbas & Pandya, 1989; Pandya & Barnes, 1987). From the limbic core of the dorsal moiety, however, emerges a positive affective bias, which motivates projective functions of the dorsal cortex. The visceral component of the dorsal stream includes most of the classic Papez circuit; subcortical structures arch from the hippocampus just lateral to midline cingulate cortex. The dorsal frontal cortex, functioning primarily to interface with the environment, also controls this limbic component by suppressing or disinhibiting the limbic bias.

Cognition from motor control in the frontal cortex. The self-regulation of motor action provides an accessible demonstration of the dynamics described above. The dorsal system provides a ballistic projection of motion (e.g., reaching for the cup of coffee), while the ventral system adapts the motion according to feedback from the environment (e.g., fine motor adjustment in response to the temperature of the mug). In this simple motor task, visceral components (i.e., limbic) provide affective biases that motivate and constrain somatic (i.e., cortical) function. These affective biases provide the motivation to act on the ballistic motor plan (positive affect) and anxious attention to cues that will guide fine motor adjustment (negative affect).

The somatic components also regulate the limbic structures, mediating the extent of emotional drive. If this reciprocal control were not in place, we would likely push the cup over with an over-enthusiastic motor plan. Or we would fail to grasp the cup altogether and instead make ongoing, obsessive and anxious fine motor adjustments. The two systems provide reciprocal control. From this description of frontal lobe self-regulation in motor action, we can imagine how complex psychological phenomena

might emerge from reciprocal control systems. It also becomes clear that cognition and emotion are not separable functions, but interdependent properties of brain function.

Having established this framework of frontal lobe self-regulation, however oversimplified, it is now possible to speculate on the neural properties of psychological phenomena. When humans report on their affective experience, or the traits of other people, those reports take on a dimensionality that resonates with these neural control processes. A bias toward one control process can be observed in complex human behavior (Tucker & Williamson, 1984). Unsurprisingly, affective biases that are fundamental to brain function manifest as trait-like tendencies in mood (i.e., positive and negative affect), personality (i.e., extraversion, neuroticism) and psychopathology (i.e., externalizing and internalizing disorders).

Depression from dysfunction in neural control systems. Frank Benson and Dietrich Blumer (1975) reviewed psychological changes associated with specific neurological impairments in frontal control systems. The authors observed that when a lesion occurs in dorsal limbic structures, symptoms relate to motivation (i.e., positive affect). In the motor system, this presents as *akinetic mutism* or *transcortical motor aphasia*. These fundamentally motor conditions also render the patient psychologically inert, exhibiting a lack of behavior activation and blunted affect. Ironically, these neurological symptoms are sometimes described as *pseudodepression* because the etiological root was neurological not “psychological.” Yet dorsal limbic hypofrontality is also a common characteristic in patients with Major Depressive Disorder, where etiology is considered psychiatric (Mayberg, 1997). This neural abnormality is also associated with motor and cognitive symptoms of depression; physical and mental slowness. Despite these shared neural origins, depression is not viewed as a neurological disorder.

When both neurological and psychiatric patients present with pathology specific to a region of the frontal cortex, the psychological profile is consistent. With damage to the ventral system, the dorsal system is under regulated. This failure to constrain the impulsive, expectant mode inherent to dorsal stream presents as disinhibitory psychopathology (Blumer & Benson, 1975). A classic case study of *disinhibition syndrome* is Phineas Gage, who suffered damage to his ventral prefrontal cortex when a tamping rod was thrown through his head (H. Damasio, Grabowski, Frank, Galaburda, &

Damasio, 1994). More focal lesions to the ventral prefrontal cortex are associated with severely decreased capacity to predict negative social consequences of actions, including a related psychiatric condition known as *pseudopsychopathic syndrome* (Blumer & Benson, 1975). Brain imaging research on psychopathy is also consistent with this view. Psychopaths appear to have a specific deficit in self-regulation, such that social information can be acquired, but it fails to elicit the appropriate adjustments of behavior (Kiehl, 2006). When the failure is in the cortical control of limbic functions in the ventral cortex, the ventral limbic structures may be disinhibited. Unsurprisingly, state, trait and pathological anxiety are associated with hyperactivity in the ventral limbic structures (Etkin & Wager, 2007; Phan Luu, Collins, & Tucker, 2000).

Depression, in contrast, is associated with dorsal hypofrontality and anxiety is associated with ventral hyperfrontality (Drevets et al., 2008; Fitzgerald et al., 2008; Mayberg, 1997). The more common clinical presentation, however, is complicated by co-morbid anxiety and depression (Kessler, Berglund, et al., 2005; Lamers et al., 2011). This is not surprising, as a control-systems model would predict compensatory reciprocity. Co-morbidity is a common obstacle to effective treatment planning for both patient and clinician. In a co-morbid presentation, it is difficult to identify the primary source of pathology, or the domain most amenable to change. Although self-report psychometrics provide a gross measure of control biases, a more direct measure of frontal lobe control systems – dorsal and ventral, somatic and limbic – might better characterize disorders of affect and direct treatment.

Self-evaluative cognition from control systems of the brain. I return now to self-evaluation, as defined by the act of self-report on basic psychometric assessments. In psychometric research, population level statistics are used to characterize patterns in human experience. An individual's aggregated scores on a psychometric scale describe their affective tendencies relative to other people. Following the discussion above, these affective traits emerge from neural control biases. Variance in trait affect might reflect variance in control system bias (i.e., the dominance of one system over another). A goal of the current research is to characterize the action of neural control systems in the development of a self-evaluative decision. One approach to this aim will be to identify neural correlates of trait affect in neural measures during self-evaluation.

A single act of self-appraisal emerges from cybernetic control processes in the frontal lobe. Using motor action regulation as a template, we can speculate on the processes that may be involved in making this decision. We might imagine a self-schema, embodied in memory, as a learned, probabilistic model. Like a ballistic motor plan, the self-schema is motivated by positive affect and thus formulated on the substrate of positive hedonic tone. An individual with pathologically low positive affect might suffer, quite literally, from an insufficient self. The word stimulus (and recognition of its meaning) must then be compared to this insufficient predictive model, or self-schema. Vigilant ventral functions are engaged to assess the stimulus relative to internal need states. Should ventral-limbic bias overwhelm the dynamic, as in high anxiety, an individual might be overly attentive to the content of the word (e.g., qualities of social desirability or valence). As the motor plan is finely adjusted according to environmental information, so the self-schema is elaborated with anxious information.

In Chapter III, I will approach this model of self-evaluative cognition from the perspective of event-related potential research. The EEG recording suggests stages of neural processing that align with the theoretical process described above. What this theoretical exercise highlights is that self, like any motor or cognitive process, is an emergent property of a self-regulating, system of systems. In this formulation, affect is not a mediator, but a substrate of the self. A core property of the brain, and thus the self, is affective.

Part III: Toward Brain-Based Metrics of Neural Self-Regulation

Hemispheric specialization: a superordinate control system. The self-regulation in the brain is achieved through reciprocal control processes in the somatic and visceral systems, and between the dorsal and ventral streams. On a more global scale, functional divisions can be made from the left and right hemisphere. Although differences can be subtle, the division of functions between hemispheres is a key adaptation in the course of human evolution; cognitive capacity is enhanced by the specialization of local circuitry, while global connectivity is retained. (Striedter, 2005). Hemispheric specialization allowed this capacity to evolve with limited metabolic cost and increase in skull diameter (Striedter, 2005).

In most humans, the left hemisphere is object oriented. It is dominant for processes related to language and speech production, in particular. This effect is robust enough that ERP experiments that involve language, including self-evaluation, will show some left lateralization on the head surface (Neville & Bavelier, 1998). The right hemisphere, in contrast, appears to be dominant in spatial operations (Weintraub & Mesulam, 1987). Of the two hemispheres the right is also more concerned with affect (Schwartz, Davidson, & Maer, 1975; Tucker, Hartry-Speiser, McDougal, Luu, & Degrandpre, 1999). Even in these hallmark domains, the functional distinction between hemispheres is less one of specialization and more of style. The left hemisphere, for example, is independently adept at solving spatial problems. Important aspects of language, such as emotional prosody, are dominated by the right hemisphere (Ross & Mesulam, 1979).

The left and right hemispheres also take on some functions of the ventral cortico-limbic and dorsal cortico-limbic system, respectively. The left hemisphere has an affective bias that is preferentially anxious (i.e., negative affect). This is consistent with left hemisphere bias for serial and object oriented processing in the ventral “what” path. The right hemisphere, in contrast, is more aligned with the positive bias of the dorsal system, which motivates the holistic processing style represented in the “where” pathway. More evidence for this asymmetric affective bias is drawn from neurological cases in which specific lesions to left and right cortices are associated with symptoms related to NA and PA, respectively (Gainotti, 1989; Robinson, Kubos, Starr, Rao, & Price, 1984).

First principals of neural self-regulation suggest that the functional dynamic between hemispheres is more competitive than collaborative. Transcallosal fibers are mostly inhibitory, suggesting that like somatic and visceral systems, as well as dorsal and ventral moieties, the hemispheres are also engaged in a dynamic of reciprocal control (Bloom & Hynd, 2005). In the current research, I anticipated some left-lateralization of the ERP in processes related to reading and semantic retrieval. Later in the decision-making epoch, I predicted some right lateralization in ERPs related to emotion processing. The affective-arousal, control-systems model also suggests that some left hemisphere functions take on an NA bias, while some right hemisphere functions take on

a PA bias. I will test this hypothesis by assessing correlations between individual differences in trait affect and neural measures in each hemisphere. If this hypothesis is confirmed, however, the finding will contradict evidence derived from research on alpha asymmetry, which predicts a different pattern of affective bias in frontal cortices.

Alpha asymmetry and mood dimensionality. Frontal asymmetry in the EEG alpha frequency band has been carefully studied, beginning with the seminal work of Richard Davidson (e.g., 1992). On the time scale of cognitive events, alpha band oscillations are relatively slow (8-12 Hz). The presence of alpha over frontal cortex reflects functional quiescence, or “deactivation,” because the subtle variations that convey information in the brain are absent. Alpha asymmetry refers to the differential in alpha power between left and right anterior channels, suggesting that one hemisphere is more active (less alpha) than the other (more alpha). Alpha asymmetry appears consistently in depressed and anxious people; greater activation is observed in the right frontal cortex, relative to the left (Thibodeau, Jorgensen, & Kim, 2006). In over 100 studies alpha asymmetry has been reliably associated with emotion and behavior (reviewed in Coan & Allen, 2004).

Alpha asymmetry is trait-like; it is relatively stable in individuals over time. An aspect of the phenomenon is heritable, and thus alpha asymmetry is considered *endophenotypic* (Stewart, Bismark, Towers, Coan, & Allen, 2010). The alpha metric not only discriminates acutely depressed from non-depressed individuals (Stewart, Coan, Towers, & Allen, 2011), but also identifies non-depressed people who are at risk for the disorder (Bruder et al., 2005). For these reasons, alpha asymmetry was a promising brain-based measure of dysfunctional emotion regulation in the frontal cortex. However, effect sizes are moderate (Thibodeau et al., 2006) and the phenomenon is not specific to mood and anxiety disorders. In clinical populations, it alpha asymmetry is instead broadly related to internalizing disorders (Stewart, Levin-Silton, Sass, Heller, & Miller, 2008). In non-clinical populations, it has been associated with withdrawal motivation, (e.g., Coan, Allen, & McKnight, 2006) and approach motivation (e.g., Davidson, 1998).

A common interpretation of these findings is that cortical activation (i.e., decreased alpha power) in the right frontal hemisphere reflects active generation of negative affect; cortical activation in the left hemisphere reflects generation of positive

affect. This is inconsistent with the control-systems hypothesis, which posits NA bias in left hemisphere and PA bias in the right hemisphere. An alternative explanation for the alpha findings is that cortical activation represents the suppression of limbic drive, not generation of affect. This explanation is more consistent with the evidence reviewed in the previous section.

Toward a neurometric of control processes in the frontal lobe. The alpha asymmetry metric has thus far fallen short as a brain-based assessment of mood dysfunction. Both its promise and limitations, however, represent a growing area of interest and discussion in clinical neuroscience (Briesemeister, Tamm, Heine, & Jacobs, 2013; Buzsaki & Watson, 2012; Miller, 2010; Siegle et al., 2012). Though currently beyond the scope of neuroscience and engineering, one can imagine a future in which *neurometrics* have been constructed for early detection and treatment of some psychological disorders. The current research was motivated by this distant goal. The methods reviewed in the chapter to follow reflect an attempt to align the analysis of neural activity with psychometric research, the discipline that has been most productive in developing measures of psychological phenomena for clinical use.

Though directed toward the development of brain-based assessment tools, the current research yields results that are more relevant to basic science. A goal of the research described in subsequent chapters is to identify neural correlates of self-appraisal bias. Findings may be theoretically informative, but a neurometric measure of positivity bias would be redundant with the behavior itself. However, there are different neural conditions that might contribute to the loss of positive bias in depression. This points the broader goal of this research: to characterize network activity in the ERP signature. Measures of network activity would not be redundant with the behavior, but might instead suggest *how* the behavior (i.e., self-evaluation) is constrained by affective control bias.

In Chapter III, I describe a methodological approach to this goal using statistical decomposition of the EEG data. I hypothesized that this strategy would separate the ERP data into functional components. Results were then interpreted within a control-systems framework of neural self-regulation. This model of frontal cortical functioning suggests several dynamic profiles that might account for the loss of positive self-appraisal bias in

depression. A broader goal for this area of research is to develop measures of network status that might differentiate these dynamic profiles of frontal lobe self-regulation.

CHAPTER III

PSYCHOMETRICS IN EEG RESEARCH

The previous chapter provided a theoretical context for this research. In Chapter III, I derive specific hypothesis from a review of the event-related potential (ERP) literature. To begin, I introduce a *neuropsychometric* approach to the analysis of brainwave data. This analytic approach convolves psychometric and ERP methods in an attempt to identify neural activity related to the dimensions of trait affect: PA and NA, respectively. I then review an application of the neuropsychometric approach to the study of self-evaluative cognition. Results of this investigation suggested two systems of cortical activity within the evaluative epoch. I conclude by discussing the limitations of the neuropsychometric approach. I note that in experimental research, affect can be operationalized in two ways; relative to stimulus attributes (e.g., word valence), or individual differences in mood state (e.g., trait PA). However, these constructs are not independent, and both must conform to a biological framework.

In the second part of Chapter III, I provide rationale for the specific methods and hypotheses tested in Chapters IV, V and VI. I begin with an introduction to principal components analysis (PCA) in psychometric and neuroimaging research. I then provide rationale for the application of PCA to ERP analysis in the study of self-evaluative cognition. A challenge in this line of research is that several of the decision-making ERPs are superimposed; they overlap in time and space. I propose that PCA will effectively separate these ERPs for a more accurate study of their unique significance. I briefly review research that has defined the functional significance of the following ERPs: P300, LPP, P1r & MFN. The current research contributes to this literature by providing a whole-brain context for these waveforms, which are typically studied in isolation.

I conclude this chapter with an introduction to three studies of self-evaluative cognition. I first introduce Study 1, which investigated self-evaluation in depression, at various levels of symptom severity. To better interpret findings in this clinical sample, I then developed a model of “neurotypical” functioning with Study 2 and Study 3. In Study 2, I introduced an “other-evaluation” contrast in an effort to isolate neural indices of self-specific processing. I also address this goal in Study 3; participants engaged in both self-evaluation and a word categorization task. To conclude, I review the central

goals of this research and introduce hypotheses that are relevant to the chapters that follow. Specific methods and results of these studies are described in Chapters IV-VI. To avoid redundancy, however, I provide a general introduction here.

Part I: A Neuropsychometric Approach

Psychometric methods in ERP research. Neural self-regulation is achieved in the brain because inter-related control systems provide reciprocal motivation and constraint (Tucker & Luu, 2012; Waters & Tucker, 2013a). Some brain functions are biased by internal need states, such as hunger or loneliness. Other systems are more responsive to the external environment; sensory experiences that are discrepant, surprising or threatening. A control-systems model of frontal lobe self-regulation posits that at the core of each of these systems is a source of affective-arousal: PA and NA, respectively.

The concepts of PA and NA, however, are derived from psychometric research. They are, by definition, latent factors that together describe much of the subjective, emotional experience (Watson & Tellegen, 1985). In research and practice, psychometric questionnaires are used to measure the status of these latent factors in individuals. For example, the Positive and Negative Affect Schedule (PANAS), presents ten words that are highly and specifically related to PA and NA, respectively (Watson et al., 1988) (Table 1). The average endorsement across each set of ten words provides an estimate of affective arousal bias in individuals; PA and NA summary scores. According to a control-systems model of neural self-regulation, this latent factor structure of mood also reflects trends in brain activity. I hypothesized that, just as word items on the PANAS questionnaire reflect PA and NA, aspects of the EEG signature could be identified as highly and specifically related to each mood dimension.

To test this hypothesis, I developed a *neuropsychometric* approach to EEG analysis in which average ERPs would reflect affective arousal bias in brain activity. I hypothesized that PA and NA would be related to dorsal cortico-limbic and ventral cortico-limbic systems, respectively. This approach captures variance in single trials that is typically lost in brain imaging research. The neuropsychometric method is among several recent developments in single trial analysis (Waters, Song, Luu, & Tucker, 2012).

Table 1

PANAS Word Items and Loading Scores on PA and NA

PANAS descriptor	Loading Scores	
	Positive Affect	Negative Affect
Enthusiastic	.75	-.12
Interested	.73	-.07
Determined	.70	-.01
Excited	.68	.00
Inspired	.67	-.02
Alert	.63	-.10
Active	.61	-.07
Strong	.60	-.15
Proud	.57	-.10
Attentive	.52	-.05
Scared	.01	.74
Afraid	.01	.70
Upset	-.12	.67
Distressed	-.16	.67
Jittery	.00	.60
Nervous	-.04	.60
Ashamed	-.12	.59
Guilty	-.06	.55
Irritable	-.14	.55
Hostile	-.07	.52

Note: Adapted from (Watson et al., 1988)

Psychometric weighting of single trials. The *neuropsychometric* approach is a combination of psychometric and event-related potential methods. Psychometrics measure a latent construct as the average of responses across several closely related items. The average score is a more stable representation of the latent construct. In ERP research, measures of EEG amplitude are also averaged over many (sometimes hundreds) of trials. The average ERP is a more stable representation of the neural response to an event because individual trials contain a lot of variation that may be unrelated to the experiment. The fluctuating voltage in an average ERP waveform represents electrical events that are time-locked to the stimulus.

ERP methodology can be even more closely aligned with psychometrics. The self-evaluation task used in the current research closely resembles a standard

psychometric assessment. On each trial, the participant is presented with a word (e.g., brave) and must rate the extent to which the word is self-descriptive. In total, 150 descriptor words were selected for the experiment. Two psychometric properties of each word stimulus were calculated using factor analysis; PA and NA loading scores. Loading scores quantify the extent to which a word item represents a latent factor (e.g., Table 1). The advantage of having this information in ERP research is that the single trial representation of PA and NA can also be quantified.

As if generating a trait affect summary score, I weighted single trials of the self-evaluation task by their loading on the dimensional constructs of interest, PA and then NA (Figure 1). This neuropsychometric technique could be used for the study of any dimensional system, including models of personality and intelligence. It is more compelling, however, to test a two-dimensional model of affective arousal (i.e., mood) because there is evidence that these constructs also reflect a fundamental aspect of brain functioning (Tucker & Luu, 2012; Waters & Tucker, 2013a).

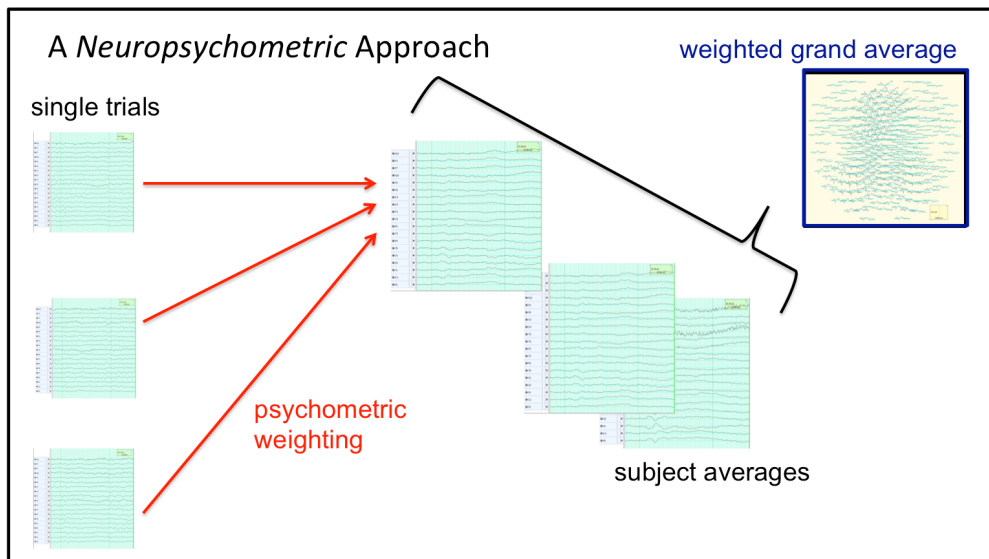


Figure 1. Schematic of neuropsychometric approach to ERP analysis. Loading scores of word stimuli are used to weight single trials of EEG data. Single subject averages are constructed from weighted, single trials. In the grand average reflects the central tendency of the psychometric construct.

Weighted averages show the time course of mood-cognition interaction. As The first demonstration of the neuropsychometric approach was conducted on a large sample of children and adolescents, ages 9-19 (Waters & Tucker, 2013b). The children engaged in a psychometric task during EEG recording. On each trial, they rated a single word adjective as *like me* or *not like me*. Responses were provided in less than one second. Importantly, each trial involved a word stimulus that was characterized with PA and NA loading scores. Single subject averages were then constructed from PA- and NA-weighted trials, such that two grand averages each reflected the central tendency of a mood dimension. The developmental nature of this sample was incidental, and may even have complicated the results. The large sample size, however, was appropriate to test a novel method.

Overall, the NA-weighted ERP was of greater amplitude (Waters & Tucker, 2013b), as if the neural mechanisms of a self-evaluative decision are dominated by NA bias. Distinction between weighted ERPs appeared 300 ms after the word was presented, coincident with the medial frontal negativity (MFN) ERP. This separation persisted through the decision-making epoch (Figure 2). This time course is consistent with research showing the interaction of emotional attributes and cognitive operations begins at later stages of the decision-making epoch (Poulsen et al., 2009; Scott, O'Connell, Leuthold, & Sereno, 2009; Tucker, Luu, Desmond, et al., 2003; L. A. Watson, Dritschel, Obonsawin, & Jentsch, 2007).

Estimating the cortical activity from PA- and NA-weighted averages. The weighted averages were further decomposed using neural source analysis, or inverse modeling. This statistical technique estimates and visualizes the cortical sources of electrical activity recorded on the scalp surface (Scherg, 1990). Source analysis provides anatomical information allowing for comparison of results with metabolic brain imaging techniques. Importantly, this transformation informs anatomical models of brain function on the timescale of cognitive events. For this analysis, I parsed the cortex into large divisions reflecting Brodmann areas. This strategy provided conservative source models that estimate cortical information within the spatial resolution of the technique.

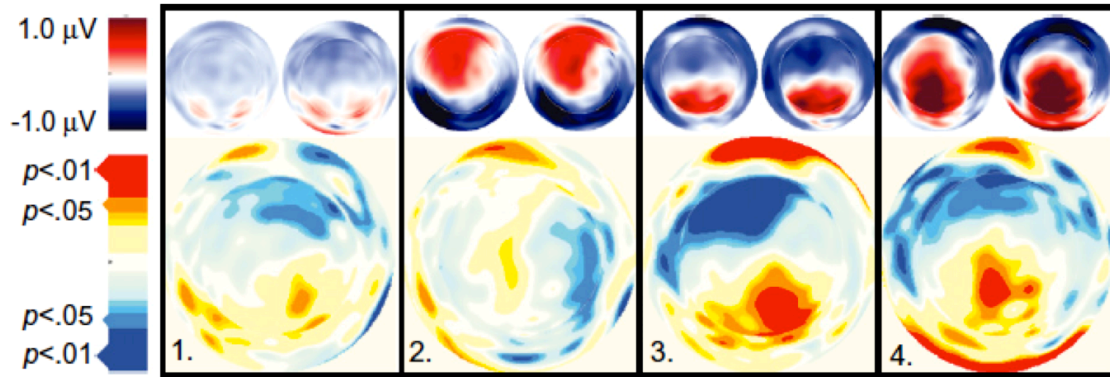


Figure 2. Topographic maps of PA- and NA-weighted ERPs and statistical difference. Topographical maps of weighted ERP amplitude and statistical difference. Left column: color scales for ERP amplitude (top) and p -values associated with t-test statistical comparison of PA- and NA-weighted average ERPs (bottom). Top row: pairs of topographical maps (PA-weighted on left, NA-weighted on right) of average ERP amplitude on the scalp surface (nose to the top of the page). Bottom row: topographic arrangement of t-statistics on the head surface showing statistical comparison of PA- and NA-weighted ERPs at each recording channel. (1) P1: 100-200 ms, (2) P2/N1: 200-250 ms, (3) MFN/P1r: 300-400 ms, (4) LPC/LIAN: 700-900 ms.

Using the neuropsychometric approach, differences in PA-and NA- weighted averages made a surprising pattern not seen on the scalp surface (Waters & Tucker, 2013b). This pattern differentiated an anterior-ventral phenomenon from a posterior-dorsal phenomenon (Figure 3). In anterior-ventral sources, PA- and NA-weighted source waveforms diverged at 400 ms and rose to a sharp peak by 850 ms. In dorsal posterior sources, the distinction began later, at 600 ms. The dorsal waveform also rose to a broad, rounded maximum by 750 ms. Although both systems showed a larger effect of NA, differences in wave shape and time course suggested complementary networks functioning within the decision making epoch. The anterior network showed effects of affect earlier than the posterior network, and showed a rapid engagement starting at 600 ms. The posterior network, in contrast, showed a gradual increase starting at 400 ms, and gradual decline from peak intensity. Though highly speculative, the two systems share attributes with fast and slow learning systems (i.e., experience dependent neural encoding) in the ventral cortical-limbic and dorsal cortico-limbic anatomy, respectively.

The neuropsychometric approach makes both theoretical and methodological contributions to the ERP literature. First, the analytic method places emphasis on

variance in single trials; it allows researchers to make use of single-trial variance within an averaged ERP design. Second, it presents the time course of neural source activity in the form of Brodmann area waveforms. This combines the temporal resolution of ERP with adequate spatial resolution to test network models. Finally, results of this initial analysis suggested a role for affective dimensions in network models of brain function. Although hypotheses were aimed at some of the more classically studied ERP waveforms, the neural source results suggest network level differentiation of PA and NA bias.

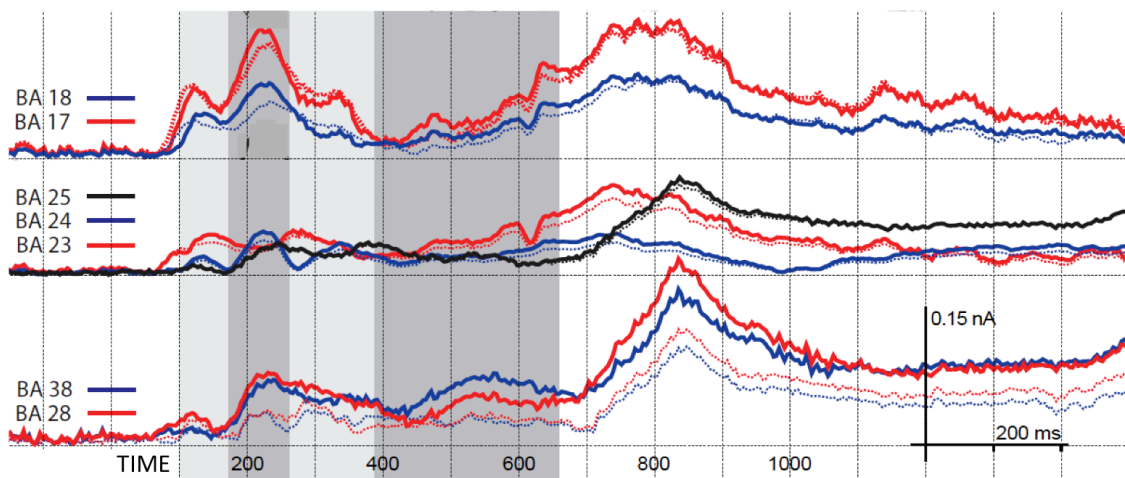


Figure 3. Source waveforms in selected Brodmann areas (BAs). NA-weighted in bold, PA-weighted are stippled.

Future directions for the neuropsychometric approach. In the neuropsychometric analysis, affective experience was operationalized relative to properties of the word stimulus. These stimulus attributes are estimated using population-level means. The technique is thus better aligned with a standard experimental design used to investigate effects of word valence (i.e., pleasant or good words; unpleasant or bad words). This dichotomous valence contrast, however, collapses all stimuli into two categories. All bad words, for example, are treated as if they are equally bad, which is not a valid assumption. For those studies aimed at better understanding the neural response to emotional stimuli, important variance is lost using this standard approach. The

neuropsychometric approach may be more precise in weighting single trials according to a quantified association with the attribute of interest (e.g., valence).

More importantly, PA and NA concepts better align experimental stimuli with constructs that occur in nature (i.e., in the brain). Although words can be understood conceptually on valence and arousal dimensions, the neural structure of mood is better aligned with the affective-arousal rotation, PA and NA. Perception of affect must conform to the perceiver's system of making meaning; there is no "bad word" without a perceiver to make the judgment. The structure of mood bias in the perceiver determines the affective qualities assigned to a stimulus. This points to what may be a conceptual limitation of both the neuropsychometric approach and the standard experimental design; both characterize stimulus attributes independent of the perceiver. In the study of mood-cognition interaction, it follows, there are at least two ways to operationalize affective experience; affective attributes of stimuli and individual differences in mood state. A future direction for the neuropsychometric approach will be to personalize weighting statistics to an individual's trait mood. A goal would be to characterize patterns of variance in single trials that differ from a population mean as a function of an individual's mood state.

In the experiments described below, both stimulus attributes (i.e., word valence) and individual differences in mood state (i.e., trait PA and NA) were applied to an investigation of mood-cognition interaction. In Study 1, variance in depressive symptom severity was used to identify neural correlates of mood-cognition interaction. Study 2 and Study 3 provided a model of "neurotypical" functioning to serve as context for dysfunctional deviation. Across all studies, I identified network features in the average ERP. In the section that follows, I describe an analytic approach to that goal using principal components analysis. I then derive specific hypotheses from a review of previous ERP research on self-evaluative cognition and decision-making.

Part II: Principal Components Analysis in Event-Related Potential Research

From average ERPs to principal components. Results from the neuropsychometric analysis suggested that the decision-making ERP could be viewed within a neural systems framework. The presence of distinct features in the source waveforms implied a ventral-anterior component and a dorsal-posterior component. To

further explore this hypothesis, I again borrowed from psychometric methodology. Factor analytic strategies have been previously used to elucidate the latent, dimensional structure of mood. I proposed that the latent factor structure in EEG might elaborate on evidence of network dynamics in the decision-making ERP. To derive latent factors from variance in EEG data, I applied principal components analysis (PCA) to the average ERP.

PCA is a statistical approach to data reduction. Variables in complex data can be summarized with a smaller number of latent factors, or principal components. Mathematically, a component is a set of weights derived from the patterns of covariance between correlated variables. Conceptually, PCA groups the variables that show similar patterns of change. For example, in the case of a 100-word survey, a subset of words shows a high level of covariance (e.g., distressed, upset, guilty). Observed together, these words represent a latent factor, which in this example is NA. In temporal PCA with ERP data, the variables are not words but time points at which a measure was taken on the scalp surface. The data is not composed of responses to the questionnaire, but samples of EEG amplitude. At each time point, covariance is derived from three sources: between subjects, between conditions and between electrodes. The latent factor structure of the average ERP provides a summary of correlated neural events.

Self-evaluative cognition in ERPs. In Chapters IV-VI, I report on three separate studies in which I used PCA to explore the latent factor structure of the self-evaluative ERP. In Study 1, I compared results obtained from depressed and non-depressed adults. In Study 2 and Study 3, I developed a neuro-typical model with data obtained from two separate samples of undergraduate participants. Each of the studies in this series provided a unique source of variance for the components analysis. In Study 1, a range of depression pathology was represented within the sample; from mild (or absent) to frequent and severe. In Study 2, participants also engaged in an other-evaluation task, wherein they evaluated a public figure. Study 3 also included a contrast condition; participants categorized the descriptor words according to their semantic meaning. To avoid redundancy in subsequent chapters, I will provide background context for this series of experiments in the sections that follow. Drawing from this literature, I present a series of hypotheses that guide the more exploratory aims of this research.

P300 and the maintenance of self-schema. The decision-making ERP can be roughly divided into a series of wave shapes. These aspects have been labeled, often according to onset time, and the behavior of each is typically studied in isolation. The first wave shapes in the decision-making epoch reflect an initial, visual response (e.g., visual P100, N1, frontal P200). This sensory response can be modulated by attention and a variety of stimulus attributes, but is relatively stereotyped (L. A. Watson et al., 2007). I instead begin this review with posterior ERPs that have been previously associated with mood, or stimulus valence (e.g., P300, LPP) (G. Hajcak, MacNamara, & Olvet, 2010). These ERPs have also been implicated in the neural basis of self (Fields & Kuperberg, 2012; Herbert, Herbert, Ethofer, & Pauli, 2011; Herbert, Pauli, & Herbert, 2011; Liu, Sheng, Woodcock, & Han, 2013; Tucker, Luu, Desmond, et al., 2003; L. A. Watson et al., 2007; Zhang, Guan, Qi, & Yang, 2013). I then review evidence that an anterior ERP, the medial frontal negativity (MFN), reflects the initial interaction of affect and cognition (Tucker, Luu, Desmond, et al., 2003; Waters & Tucker, 2013b; L. A. Watson et al., 2007).

Many average ERPs appear distinct, but actually reflect the sum of constituent waveforms; a superimposition of different neural events. One such ERP appears in posterior channels, starting at 150 ms in the decision-making ERP. This “posterior positivity” represents the superimposition of at least three ERP events: P1 reprise (P1r), P300 and the parietal slow wave, or late positive potential (LPP). A combination of these, typically P300 and LPP are also described as the late positive complex (LPC). Previous research has explored the behavior of these ERPs in the context of emotional decision-making (G. Hajcak et al., 2010).

The P300 is generally associated with the motivational salience of an event, as well as memory encoding (Donchin & Coles, 1988; J Polich, 2007). Early investigations showed that the P300 is enhanced when target stimuli are detected within a series of non-targets (Courchesne, Hillyard, & Galambos, 1975). The P300 is also enhanced for emotional stimuli, such as frightening scenes or joyful faces (Lang, Nelson, & Collins, 1990). These effects only occur, however, when a person is paying attention to the task (Duncan-Johnson & Donchin, 1977). Furthermore, the stimulus must carry information that is relevant to one’s motivation for attending (e.g., target detection). In other words,

the P300 response occurs within the context of an initial motivational bias (Bradley et al., 2003).

The P300 is commonly associated with memory encoding because it is enhanced following stimuli that are later remembered (Donchin & Coles, 1988). However, ERP research shows that cognitive operations are spread out in time. Thus, cognition is redefined as an ongoing process instead of a discrete event. Similarly, neural encoding associated with the P300 is better understood within a cognitive operation; one that begins with a predictive schema, and ends with adjustments to that schema. Donchin and Coles (1988), described these adjustments associated with the P300 as context updating. In the context of self-evaluative decisions, it may be an aspect of the self-schema that is updated following appraisal events. Indeed, the LPC is larger when people evaluate themselves or a close friend, compared with reading (Poulsen et al., 2009; Tucker, Luu, Desmond, et al., 2003). The LPC is also larger when a trait word is endorsed as *like me*, rather than rejected, particularly when the word reflects a socially desirable characteristic (Poulsen et al., 2009; L. A. Watson et al., 2007). This suggests that P300 encoding might be related to the positive self-evaluation bias that is consistently demonstrated in self-report measures.

Neural schema, memories or context-updating all reflect experience dependent learning. The P300 appears most related to the slow learning system of the dorsal cortex (Waters & Tucker, 2012). The dorsal limbic core is centered on the hippocampus and includes the classic Papez circuit, which includes posterior cingulate cortex (PCC). Some evidence links the P300 to activity in the PCC (Waters & Tucker, 2013b), as well as the phasic action of the norepinephrine system that innervates that region (Nieuwenhuis, Aston-Jones, & Cohen, 2005). Abnormalities in these aspects of the dorsal system, including attenuated P300 amplitude, are associated with depression and various other psychiatric disorders (Polich, 1998; Poulsen et al., 2009). Notably, P300 reactivity is relatively stable across time, suggesting that this ERP indexes trait-like aspects of the decision-making ERP (Walhovd, Rosquist, & Fjell, 2008).

The LPP and motivation to self-regulate. The late positive potential (LPP) follows the classic P300 in the posterior positivity. Depending on the experimental design, the LPP can endure for seconds beyond the decision-making epoch. The LPP is

larger following emotional or arousing stimuli, relative to neutral stimuli (MacNamara, Foti, & Hajcak, 2009). Like the P300, the LPP is stable in individuals over multiple recordings and there is some evidence that LPP reflects trait-like individual differences in motivation bias (Codispoti, Ferrari, & Bradley, 2007). For example, LPP is larger for objects that an individual desires, including those related to addiction in persons diagnosed with substance abuse. Individuals with a specific phobia show an enhanced LPP when exposed to the object of their fear, (G. Hajcak et al., 2010). Unlike the P300, notably, the LPP does not habituate after multiple presentations of a stimulus (G. Hajcak & Nieuwenhuis, 2006). This suggests that the motivational bias represented in the LPP may be more general, and less tied to a particular event than the P300.

When a stimulus is rapidly masked, the stereotyped visual ERPs remain intact, while the LPP is nearly absent (Whalen et al., 1998). This suggests that the LPP is present with conscious awareness of an event. Consistent with this observation, the LPP is attenuated when a person attempts to decrease a negative emotional response (Moser, Hajcak, Bukay, & Simons, 2006). Furthermore, self-reported changes in mood following intentional emotion regulation correlates with moderation of the LPP (G. Hajcak & Nieuwenhuis, 2006). Given this evidence, LPP is thought to index intentional and automatic emotion regulation, in the context of trait-like motivational biases.

The P1 reprise is involved in re-entrant processing of a visual stimulus. A less studied feature of posterior positivity is its earliest aspect, the P1r, which peaks between 250 and 400 ms following stimulus onset. The P1r appears as bilateral foci, a topography that resembles the P100, which is an aspect of the early visual response (Tucker, Luu, Desmond, et al., 2003). This “reprise” of the visual P100 may provide some information about the functional significance of the P1r. The time course of the P1r is coincident with the MFN, which appears in anterior channels and has been implicated in complex processing of a visual stimulus. Evidence from this topography and time course combined suggests that the P1r is an index of re-entrant visual processing that follows the engagement of frontal lobe. The P1r is sometimes right lateralized in emotional tasks, including those that engage specialized functions of the left hemisphere, such as reading (Waters & Tucker, 2013b). The P1r may also reflect additional processing of emotional information, a specialization of the right hemisphere.

The MFN detects events that are discrepant with predictions. The medial frontal negativity (MFN) is among a suite of negative going ERP components that emerge from activity in medial frontal cortices. The medial negativities reflect an exaggerated fronto-limbic response to a salient or discrepant event, self-observed errors or negative feedback, including negative self-evaluation (P. Luu, Flaisch, & Tucker, 2000; Tucker, Luu, Desmond, et al., 2003). The MFN appears in anterior channels between 250-450 ms, coincident with the P1r waveform in posterior channels.

The MFN is consistently identified as the first appearance of emotion-cognition interaction (Fields & Kuperberg, 2012), including divergent effects of mood dimensionality (Waters & Tucker, 2013b). Tucker et al. (2003) provided a detailed description of effects in medial frontal electrodes during self-evaluation. Authors reported that by 300 ms, the MFN was greater in response to bad words than good word, particularly over the right frontal lobe. Within 50 ms of this valence effect, there was an interaction of valence and endorsement, such that the MFN was largest for negative-self appraisal events. This is the earliest reported effect of endorsement, suggesting that the appraisal decision is made (though not executed) within the MFN epoch.

Importantly this enhancement of the MFN on negative appraisal trials was observed during self-evaluation, but not during the evaluation of a close friend (Tucker, Luu, Desmond, et al., 2003). It is as if the negative self-appraisal event is sufficiently salient in reference to the self and dissonant with internal self-schema. The MFN may reflect adaptive self-regulatory functions of the frontal limbic-cortex that are biased by negative affect (Tucker & Luu, 2007). However, the MFN appears exaggerated in people with high anxiety and trait negative affect (P. Luu et al., 2000). In clinical populations, the negative bias indexed in MFN reactivity might represent a disruption of implicit forms of self-regulation that contributes to maladaptive thinking.

Exaggerated MFN in negative-self appraisal trials was not, however, observed in depressed individuals (Poulsen et al., 2009). Instead, the MFN was attenuated when bad trait words were endorsed, relative to endorsed good words. The authors suggest that in depression negative self-appraisal may be congruent, not discrepant, with dysfunctional expectations (i.e., self-schema). Exaggerated MFN has also been shown in moderate depression following negative feedback, which is aligned with the experience of negative

self-appraisal (Tucker, Luu, Frishkoff, Quiring, & Poulsen, 2003). Importantly, this effect attenuated as symptom severity increased, suggesting that the frontal lobe mechanisms of self-regulation might show a non-linear relationship with symptom severity.

In summary, exaggeration in the MFN can be associated with an anxious, negative affective bias. In severe depression, the MFN response attenuates with symptom severity. Attenuation in the posterior positivity, particularly the P300, can be associated with depression (low PA). It is possible that the MFN decreases in severe depression as co-morbid symptoms of anxiety fade. It may be that co-morbid anxiety in mild to moderate depression is an adaptive response to hypoactivity reflected in the P300. Testing this hypothesis may require methods of analysis that assess the relationship between anterior and posterior ERPs. Although frontal lobe mechanisms are more directly associated with self-regulation, the study of emotional decision-making with ERP typically reflects posterior brain responses. In the following section, I introduce three studies designed to characterize covariance between ERP waveforms over both anterior and posterior cortices. This series of experiments investigates evidence of network reciprocity in latent structure of the self-evaluative ERP.

Part III: Introduction to Study 1, 2 and 3

Self-evaluative cognition in mild, moderate and severe depression. Depression involves dysfunctional mood-cognition interaction that can be observed as low trait positive affect and a marked decline in positive self-evaluation bias. In Study 1, I investigated neural mechanisms of self-evaluative cognition as a function of this decline. Results were interpreted relative to a control-systems framework of neural self-regulation. The sample included adult participants who reported symptoms of depression ranging from absent to severe. EEG was recorded while these depressed and non-depressed persons engaged in a self-evaluation task; rating single word adjectives as *like me*, or *not like me*. As expected, positive self-evaluation bias was inversely correlated with depression severity. The effect was greatest, however, in a subgroup of moderate to severely depressed individuals. This finding suggested that as depression grows more severe, negative self-evaluative cognition is more closely tied to an individual's symptom profile.

A secondary goal of this research, it follows, was to identify frontal lobe activity that showed a non-linear relationship with negative self-evaluation and symptom severity. I proposed a theoretical hypothesis that as depression grows more severe, there may be a categorical shift in the self-regulatory dynamic of the frontal lobe. In this sense, depression is distinct from transient sadness, which may be adaptive following loss. I investigated this hypothesis by first developing models of neural source activity in selected areas of the cortex (i.e., Brodmann areas). I then conducted an exploratory correlation analysis between neural source intensity, a measure of symptom severity and a measure of self-evaluation bias. Using this approach, I also addressed a tertiary aim of this study; to test the hypothesis that neural correlates of PA are right lateralized, while correlates of NA are left lateralized.

The central line of inquiry in Study 1 involved the application of principal components analysis to the decision-making ERP. I hypothesized that the latent factor structure might capture features of affect biased, reciprocal control systems in the frontal lobe. More specifically, I predicted that factors might recreate dorsal-posterior and ventral-anterior networks evidenced using a neuropsychometric approach. As this was a novel application of PCA methodology, Study 1 analyses were guided by the ERP features previously identified as relevant to affect-cognition interaction. For example, I hypothesized that the PCA would deconstruct the posterior positivity into three factors (P1r, P300, LPC) and predicted that latent factor associated with the P300 ERP would be attenuated in depressed participants.

Neurotypical self-evaluation. Both the *neuropsychometric* analysis and Study 1 involved special populations (i.e., developmental; clinical). With Study 2 and Study 3, I sought to characterize effects in a “neurotypical” sample, screened for anxiety and mood disorders. This effort was undertaken to provide a necessary contrast with clinical or developmental samples. The central line of inquiry in Study 2 and Study 3 was an attempt to replicate the time course and topography of the component structure observed in Study 1. I further hypothesized that the latent factors have a functional significance relative an affective-arousal model of neural self-regulation.

To explore this hypothesis, I developed two experimental contrasts. The implementation of contrast conditions also addressed a limitation of the Study 1.

Although I identified neural correlates of evaluation bias in Study 1, I could not conclude that these processes were specific to self-focused cognition. In Study 2 and Study 3, I address this limitation with the addition of two contrast conditions. In Study 2, I adapted the self-evaluation task to include a second condition in which participants evaluated a public figure. In Study 3, participants engaged in self-evaluation as well as a semantic categorization task. The addition of these contrast conditions was an effort to isolate the neural activity that is most directly related to self-processing.

Self-specific neural processing has been investigated with ERP methodology using a variety of experimental strategies. A frequent approach involves reading pronoun-noun pairs that indicate whether a given stimulus is self-relevant, or relevant to someone else (Herbert, Herbert, et al., 2011; Herbert, Pauli, et al., 2011; Walla, Duregger, Greiner, Thurner, & Ehrenberger, 2008; Zhou et al., 2013). Another non-evaluative approach to self-reference involved cued recall of self-specific memories (Magno & Allan, 2007). One study used a self-evaluation task that involved reading phrases (Fields & Kuperberg, 2012). At least two studies measure self-processing in the time frequency domain, instead of with an ERP approach (Knyazev, Savostyanov, Volf, Liou, & Bocharov, 2012; Mu & Han, 2010). Several studies are aligned with the current experimental design, but do not include a contrast of self- and other-reference (Poulsen et al., 2009; Waters & Tucker, 2013b; L. A. Watson et al., 2007; Zhang et al., 2013).

To the best of my knowledge, only three ERP studies have contrasted self-evaluation with other-evaluation (Esslen, Metzler, Pascual-Marqui, & Jancke, 2008; Liu et al., 2013; Tucker, Luu, Desmond, et al., 2003). This is surprising given the productive and growing use of this paradigm in functional magnetic resonance imaging (fMRI) research. Unlike the ERP literature, metabolic measures of self-processing have coalesced on a network of brain areas, particularly along the midline, that consistently differentiate self- from other-evaluation (Craig et al., 1999; D'Argembeau et al., 2005; Heatherton et al., 2006; Johnson et al., 2002; Kelley et al., 2002; Kennedy & Courchesne, 2008; Ochsner et al., 2005; Pauly, Finkelmeyer, Schneider, & Habel, 2013; Pfeifer, Lieberman, & Dapretto, 2007; Sarsam et al., 2013; Seger, Stone, & Keenan, 2004). These include the posterior cingulate and medial parietal lobe; ventral and dorsal medial prefrontal cortex. Notably, these regions also contribute source electrical activity to the

MFN and posterior positivity on the scalp surface. However the timescale of ERP and fMRI effects does not align. Whereas midline ERPs appear before 500 ms, metabolic effects are measured on the timescale of seconds.

Both Esslen et al., (2008) and Liu et al., (2013) reported a difference between self- and other-evaluation related to dorsal-medial prefrontal cortex (self>other). Esslen et al. (2008), derived this result by averaging the ERP over 700 ms of the decision-making epoch. Liu et al., (2013) report their finding specific to the LPP time window in anterior channels (500-700 ms). Tucker et al., (2003) also report an effect of reference along central midline channels as the MFN transitioned into an anterior positivity (475-525 ms). This effect reflected greater magnitude for the negative self-appraisal in self-reference, but not other reference. Both Esslen et al., (2008) and Tucker et al., (2003) asked participants to select a close friend for the other-evaluation condition. In Liu et al., (2013), participants evaluated a famous athlete. Previous studies have used political figures (Craig et al., 1999; Kelley et al., 2002; Sarsam et al., 2013). Seeking to replicate the robust frontal effect reported in two of the three previous studies, I developed a contrast for Study 2 that involved the evaluation of a distant other, President Barack Obama.

In an attempt to further exaggerate self-reflection in the decision-making epoch in Study 2 and Study 3, I also adapted the response options. In Study 1, participants had a dichotomous response option (*like me; not like me*) and the decision-making task was speeded; participants would receive a warning if responses were slower than 800 ms. In Study 2 and Study 3, I expanded the response options to a four-point scale. I also cued responses 1 s. after the presentation of the word stimulation. This forced lengthening of response times, and opportunity for more nuanced responding, was intended to intensify self-reflective processing in the decision-making epoch.

For Study 3, I developed a novel contrast condition in which participants engaged in a semantic categorization task. This task was identical to the self-evaluation task, in all ways but instruction. Participants instead categorized the word stimuli as either an emotion descriptor or a personality trait. Emotion and personality were opposite poles of a bipolar continuum so that response options would involve a four-point scale, as in the self-evaluation condition. The categorization task in Study 3 was designed as an

alternative to other “semantic” control conditions, such as counting letters in the word or identifying the font case (e.g., Kelley et al., 2002). These controls are problematic for two reasons. First, responses are dichotomous and potentially more automatic than those provided in the self-evaluation task. Second, when performing these tasks, participants do not process semantic information. A third type of “semantic” control task asks participants to rate the valence or pleasantness of a word stimulus (e.g., Liu et al., 2013). When later asked to describe their strategy, many participants report that their valence judgment was made with implicit social comparison. The categorization task, instead, maintains the semantic and evaluative processes, but evokes less social comparison.

Mood dimensionality in self-appraisal behavior. The model of neural self-regulation proposed by Tucker & Luu (2012) posits two affective-arousal biased control systems. Functions of the dorsal corticolimbic system are motivated by positive affect; functions of the ventral corticolimbic system by negative affect. Complex human behavior, including cognition, emerges from the reciprocal dynamic of these control systems. Stable biases (i.e., subtle dominance of one system over another) in neural behavior contributes to trait-like characteristics. Individual differences in trait mood, it follows, reflects this neural organization.

Poulsen et al., (2009), observed that trait PA and trait NA were uniquely predictive of self-evaluative behavior in a sample of depressed and non-depressed adults. PA predicted endorsement patterns in response to good words, only. NA predicted responses to bad words, only. The neuropsychometric sample also showed this dimensional specificity in evaluative behavior (Waters & Tucker, 2013b). The finding, and its replication, was interpreted as evidence for dimensional specificity in mood constraint on self-appraisal behavior. The broader implication is that depression (i.e., low PA) is associated with the rejection of good things, while anxiety (i.e., high NA) is associated with the acceptance of bad things. Symptom profiles are consistent with this hypothesis. Depressive behavior is characterized by apathy and inaction, while anxious behavior involves hyper-attentiveness to threats (American Psychiatric Association, 1994).

Across all studies in the current research, I tested the hypothesis that mood constraint on self-evaluative cognition shows dimensional specificity. In parallel with this

behavioral hypothesis, I explored neural dimensionality in the self-evaluation ERP. Overall, this research considered how latent factors in EEG data might reflect network dynamics in the brain. Each study in the series modulated the factor structure with a unique source of variance. In Chapter IV-VI, I outline methods and analyses and provide results. Each chapter begins with a brief overview of the rationale and hypotheses and ends with a brief summary of results, specific to each study.

Beginning with Chapter IV, I review a study that captured variance in depressive symptom severity. This variance was used to identify network functions disrupted by a loss of positive affect. In Chapter V, I review a study that tested the hypotheses in a non-clinical sample. This study also included a contrast between self- and other-evaluation. I used this contrast to identify features of the component structure enhanced by self-focus. In Chapter VI, I review a third replication of the factor analysis. This study introduces a second contrast condition that involved categorization of word stimuli. This contrast was used to identify factors that are modulated by the social context of evaluative decisions. Chapter VII provides a synthesis of the results across all studies, including the neuropsychometric approach, within a control-systems framework of neural self-regulation. I conclude with speculation on the functional nature of latent components in the self-evaluative ERP, as well as the application of neural network measures to the study of psychopathology.

CHAPTER IV

SELF-EVALUATION IN DEPRESSION

A transient decrease in positive self-appraisal may co-occur with sad mood (J. D. Brown & Mankowski, 1993; Mor & Winquist, 2002). In clinical depression this decrease is lasting and exaggerated (Rimes & Watkins, 2005). The act of self-evaluation engages frontal cortico-limbic mechanisms of emotion regulation, but it remains unclear how these normal constraints on cognition become pathological in depression. In the analysis that follows, I investigated changes in neural electrophysiology during self-evaluation as a function of symptom severity. The goal of this research was to elaborate on a control-systems model of frontal lobe dysfunction in depression.

I constructed event-related potentials from EEG that was recorded as depressed and non-depressed individuals endorsed (or rejected) adjectives as self-descriptive. I hypothesized that participants would show a positive self-evaluation bias, but that this bias would be reduced in depression. I also tested a dimensional specificity hypothesis that posits trait positive and negative affect to be uniquely predictive of responses to socially desirable and undesirable words, respectively.

To test a model of network dysfunction in the cortex, I decomposed the average ERP into latent factors, using principal components analysis (PCA). I also used neural source analysis to estimate electrical activity in selected Brodmann areas of the cortex. I then identified neural correlates of self-appraisal bias and depressive symptom severity. I also divided the sample into two groups, based on high and low symptom profiles, and identified between group differences in the EEG signature.

The factor structure of the self-evaluative ERP has not been well characterized and the functional significance of latent factors is unknown. I hypothesized that the factor analysis would separate superimposed ERPs over the posterior cortex into three constituent waveforms. Following results of the neuropsychometric study (Waters & Tucker, 2013b), I predicted that the component waveforms would separate features of a dorsal-posterior and ventral-anterior network. Consistent with previous research (Polich, 1998), I further hypothesized that the component associated with the classic P300 ERP would be attenuated in depression.

Methods and Materials

The experiment was previously conducted at Electrical Geodesics, Inc. and the University of Oregon. Investigators generously made the raw data available for further analysis.

Participants. Participants ($N=150$) were recruited from the communities of Eugene and Springfield, Oregon. Subsequent analyses excluded participants with active substance abuse or dependence, as well as previous or current experience of psychotic symptoms, bipolar disorder, or neurological disorders. Participants were also excluded who reported current use of medication for the treatment of a mood or anxiety disorder. In total, 21 participants were excluded from the subsequent analysis. Inclusionary criteria included corrected to normal vision and English language fluency. Participants received \$30 in remuneration.

Procedure. The study was conducted according to protocol and practice approved by the institutional review boards of Electrical Geodesics, Inc. and the University of Oregon. Each participant provided informed consent and then completed a series of questionnaires, including: Beck Depression Inventory; BDI (A. Beck, Steer, & Brown, 1996), Hamilton Depression Inventory; HDI (Williams, 1988), and the Oregon Self Concept Inventory (Tucker, Luu, Desmond, et al., 2003). EEG data were recorded while participants performed a self-evaluation task. The experimental session was completed in approximately two hours.

Experimental task. The self-evaluation task required participants to read a single word adjective, such as brave or guilty, on a computer screen and indicate whether or not the word was self-descriptive (Figure 4). The 209 word stimuli represented both socially desirable and undesirable personality characteristics (i.e., good words and bad words). The participant provided their answer by pressing a button with their right or left index finger (counterbalanced across participants) to indicate whether the trait is *like me* or *not like me*. A single trial of the self-evaluation task proceeded as follows: fixation cross hatch (“+”) (2000 ms), word stimulus (500 ms), blank screen (1000 ms *or when response is given*). Word presentation was grouped in four blocks. Task stimuli and response recording was controlled by E-Prime Software, Version 1.2.1.795 (Psychology Software Pittsburgh, PA).

EEG preprocessing. The EEG recording was made through a Net Amps 200 amplifier and Net Station software (Electrical Geodesics, Eugene, Or). The data was segmented into 1200 ms epochs, including a 200 ms pre-stimulus baseline. Non-EEG artifacts were detected and removed from the analysis using the Fully Automated Statistical Thresholding for EEG artifact Rejection (H. Nolan, R. Whelan, & R. B. Reilly, 2010). Single subject averages were re-referenced to the average reference. A grand average was also constructed for descriptive analyses.

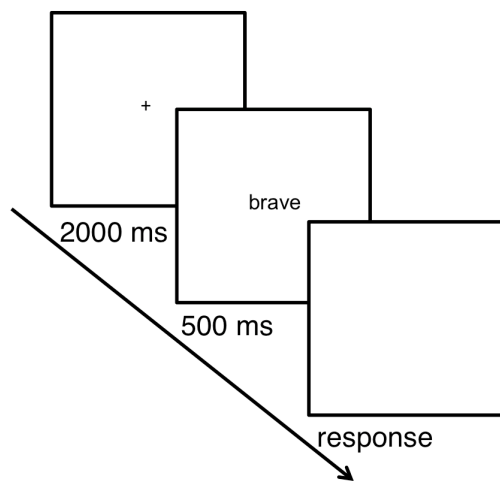


Figure 4. Stimulus presentation in Study 1.

Statistical Decomposition

Principal components analysis (PCA). The PCA was conducted with the ERP Toolkit, version 2.32 (Dien, 2010), using all single subject averages ($n=128$). A Promax rotation was used with a covariance relationship matrix and a Kaiser weighting. In this “temporal” PCA, time points are variables, analogous to items on a psychometric survey. Following decomposition into components, data were transformed back to the original scale (i.e., micro volts) by multiplying factor scores by the factor loadings for each time point (by channel by subject). The resulting waveform and scalp topography reflects the portion of the single subject ERP accounted for by each component.

Neural source analysis. A second decomposition strategy modeled electrical activity in selected cortical regions. Cortical tissue volume and location were estimated using the Montreal Neurologic Institute (MNI) MRI atlas. Skull conductivity was

estimated from an X-ray computed tomography (CT) image that was registered with the MNI atlas. A finite difference forward model (FDM) of head shape and conductivity allows cortical grey matter to be parsed into 7 mm “voxels,” or electrical dipoles (GeoSource 2.0 software; EGI, Eugene, OR). Current density in a single dipole was represented as the sum of vectors for three dimensions. Standardized low resolution brain electromagnetic tomography (sLORETA) constrained projections in the lead field. This parameter choice, coupled with a strong regularization constant (Tikhonov: 1×10^{-2}), represented a conservative (i.e., low-resolution) approach to source localization. Regions of interest were developed by grouping dipoles in large swaths of cortex. These “source montages” corresponded to twenty-three Brodmann areas (BAs) per hemisphere.

Statistical Analyses

Group differences in demographic and psychometric measures. I calculated a Percent Negative Self-Evaluation (PNSE) statistic to reflect the percent of trials in which the participant endorsed an undesirable word as *like me*, or rejected a desirable word. I used Pearson’s correlation analysis to assess the linear relationship between PNSE and BDI scores. When necessary, BDI and PNSE scores were transformed to normal. I also used one-way Analysis of Variance (ANOVA) to determine statistical differences between high BDI ($BDI \geq 10$) and low BDI ($BDI < 10$) groups on a number of variables: BDI, HDI, PNSE, age, and years of education.

Group differences in EEG amplitude. I used one-way Analysis of Variance (ANOVA) to assess statistical differences in mean amplitude between high BDI ($BDI \geq 10$) and low BDI ($BDI < 10$) groups. The ANOVA compared mean amplitude values at the peak time point of each component. No statistical correction was made for multiple comparisons. Instead, the coherent spatial organization of effects observed on the head surface is a guide to effects that survive multiple comparisons (Tucker, Liotti, Potts, Russell, & Posner, 1994).

Correlation analysis. I conducted a series of correlation analyses to characterized linear associations between BDI scores, appraisal behavior and source intensity in 46 BAs. Normality of all variables was assessed using a Shapiro-Wilk test. Non-normal variables were transformed to normal and assessed with a parametric correlation strategy (Pearson’s *r*). In some cases, non-parametric correlation strategies were used (i.e.,

Spearman's Rho). Correlation analyses were repeated on the sample divided into two groups (i.e., high BDI, low BDI). Statistical significance of the correlation coefficient was assessed using a t-distribution. In this, the t-statistic is calculated as the ratio of the correlation coefficient to the standard error of the coefficient in the sample. The null hypothesis states that the correlation coefficient is not statistically different from zero. A post-hoc analysis also tested the significance of non-linear trends in two pairs of variables. One-way ANOVA was used to test for group mean differences in two BA sources.

Results

Behavioral and demographic characteristics. BDI and PNSE appear positively correlated in a non-parametric test, $r_s(129) = .585$, $p < .001$, and when transformed to normal $r = .605$, $p < .001$. An inflection point observed in the scatterplot instructed the subsequent between groups analysis. A quadratic model added to this prediction (Figure 5), $R^2 = .417$, $\Delta R^2 = .06$, $F(2, 126) = 46.7$, $p < .001$, 95% CI [.29, .54].

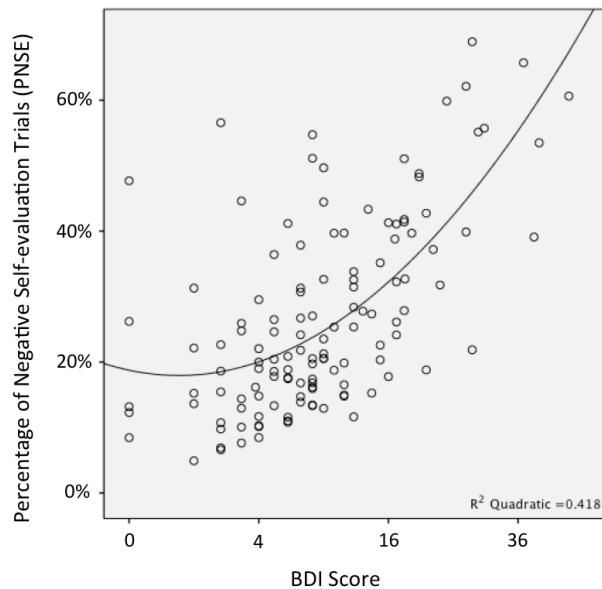


Figure 5. Correlation of % negative self-evaluation and scores on the Beck Depression Inventory (BDI).

Participants were divided into two groups, high BDI ($BDI \geq 10$) and low BDI ($BDI < 10$). Of 129 participants included in the analysis, 51 (24 male) were classified in as high BDI and 78 were classified as low BDI (32 male). Groups were statistically

equivalent in age, $p=.913$ and years of education, $p=.157$. On average, individuals in the high BDI group engaged in more negative self-evaluation (35.5%), than did individuals in the low BDI group (21.3%). The high BDI group also reported greater symptom severity on the HDI, $F(1, 127)=139.66, p<.001$. A one-way ANOVA conducted on transformed-to-normal PNSE scores confirmed a statistical difference between group means, $F(1,127)=39.761, p<.001$. A main effect of valence was also significant, $F(1,127)=9.34, p=.003$. Overall, more yes-to-bad trials contributed to the negative self-evaluation statistic. An interaction of group and valence was not significant.

Table 2

Demographic and Clinical Characteristics in Study 1

Measure	High BDI ($n=51$)		Low BDI ($n=78$)		df	F / χ^2	p
	M	SD	M	SD			
BDI	18.91	8.4	4.98	2.78	1, 127	184.95	<.001
HDI	21.12	8.45	7.73	4.25	1, 127	139.66	<.001
Age	26.08	9.17	25.88	10.22	1, 127	.012	<i>ns</i>
Education	14.6	2.06	15.27	2.86	1, 127	2.031	<i>ns</i>
PPSE	35.5%	14.6	21.3%	11.7	1, 127	37.17	<.001
Gender	24 Male		32 Male		3	.457	<i>ns</i>

Note. Beck Depression Inventory (BDI), Hamilton Depression Inventory (HDI), Age (in years), Education (in years), Percent Positive Self-Evaluation (PPSE) reflects the frequency of endorsed good words and rejected bad words. Between groups statistical assessment: One way ANOVA (BDI, HDI, Age, Education, PPSE), Pearson chi-square test (Gender). Not significant (*ns*, $p>.05$).

Means (with standard deviations) for trait positive and negative affect in the low BDI group were 37(5), and 19(6), respectively. Means for trait positive and negative affect in the high BDI group were 33(7), and 24(7), respectively. PA was positively correlated with the endorsement of good words and bad words, $r_s(129)=.523, p<.001$; $r_s(129)=.185, p=.032$, respectively. NA was inversely correlated with the endorsement of good words and bad words, $r_s(129)=-.327, p<.001$; $r_s(129)=-.531, p<.001$, respectively (Table 3).

Table 3

Correlation of Trait Affect and Self-Evaluative Behavior in Study 1

Individual Difference Measure	1	2	3
1. Positive Affect (PA)	--		
2. Negative Affect (NA)	-.175*	--	
3. Percent <i>yes</i> to good words	.523**	-.327**	--
4. Percent <i>no</i> to bad words	.185*	-.531**	.795**

n=129 **p*<.05, ***p*<.001

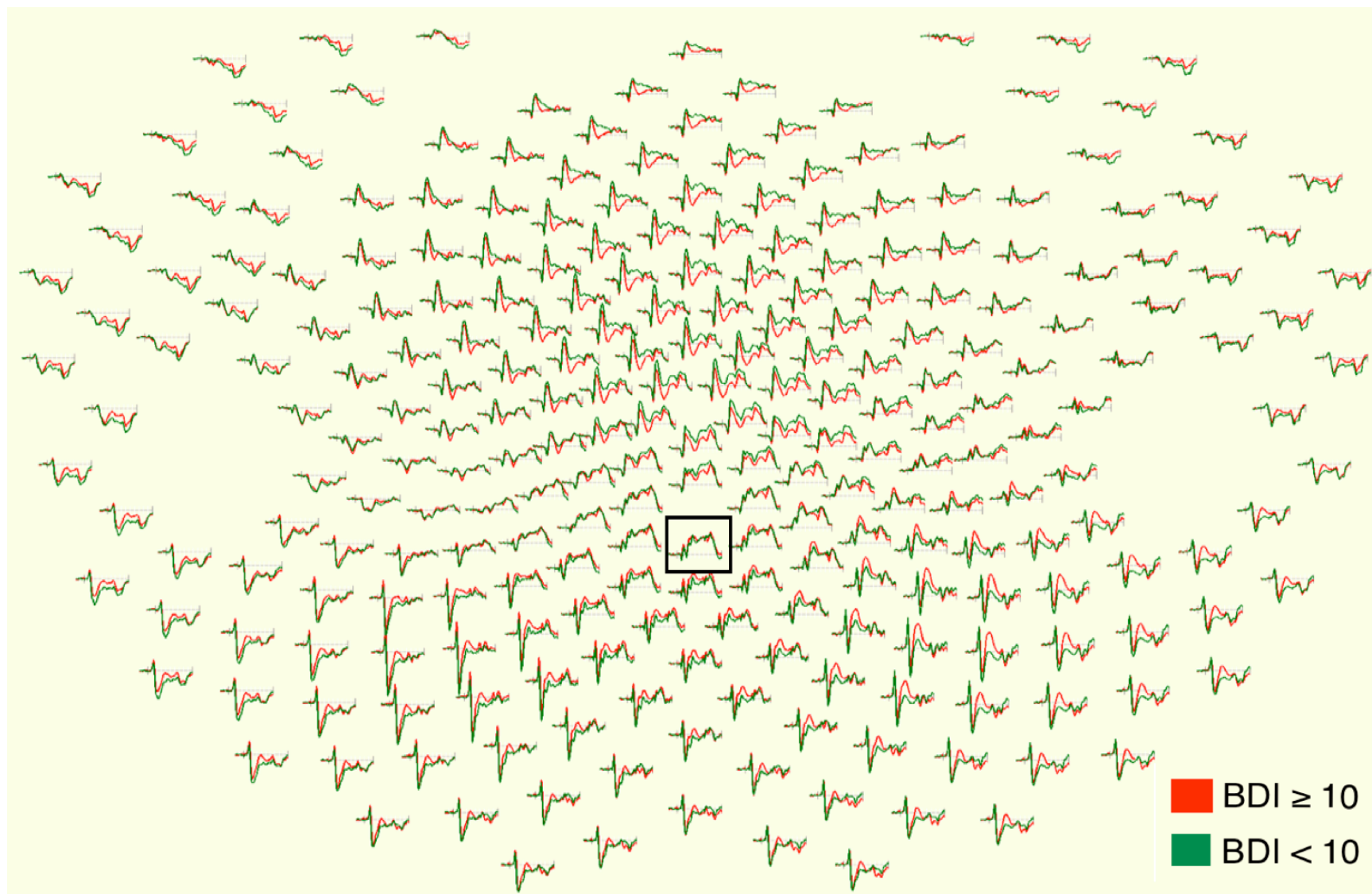
Average event-related potential. The visual P1 appears as bilateral foci and broadly across posterior channels between 100-150 ms following stimulus onset (Figure 6). It peaks at approximately 130 ms and appears greater in channels over the right hemisphere. The visual N1 appears as bilateral foci and broadly across posterior channels between 150-260 ms following stimulus onset. Amplitude reaches a minimum at approximately 185 ms. The frontal P2 appears between 155-300 ms following stimulus onset. It peaks at approximately 210 ms. The frontal P2 is highly left lateralized in this sample, extending into channels over the left temple. The medial frontal negativity (MFN) appears between 300 and 460 ms, also left lateralized in anterior channels. Amplitude reaches a minimum at approximately 360 ms. The magnitude of the MFN appears larger in the high BDI group.

The late positive complex (LPC) appears over parietal cortices between 240 and 825 ms. Visual inspection of the LPC waveform suggests three overlapping components. The first could be described as the posterior P2 or a P1r. It appears between 240-400 ms following stimulus onset with bilateral topography similar to the P1. It is strong over right parietal channels, particularly for the high BDI group, and is nearly absent over the left hemisphere. A second feature of the LPC appears between 400-680 ms. Its posterior parietal topography is roughly consistent with the classic P300 ERP. A third feature of the LPC, most consistent with the late positive potential (LPP), appears between 680-825 ms. It is distributed broadly over the parietal lobes, and reaches peak amplitude at 740 ms following stimulus onset. Figure 6 shows the average ERP for both high BDI and low BDI groups.

Latent factor structure of the average ERP. Based on the results of a parallel test, seven components were retained from the ERP data, accounting for 89% of the total variance. Components 2-4 (C2-C4) accounted for three aspects of the late positive complex (LPC), each with distinct time course and topography (Figure 7). C2-C4 accounted for 24%, 18% and 16% of the total variance, respectively. The remaining four components accounted for 13%, 8%, 6% and 4% of the total variance, respectively.

C3 is the largest component (24% of the variance). Time samples that loaded highly on this component fell between 300-760 ms following stimulus onset. The component reached peak magnitude at 540 ms. The topography of C3 reflected features of the classic P300 positivity in posterior channels, in addition to a right lateralized frontal positivity. C2 (18% of variance) described an earlier component with prominent features between 190-540 ms following stimulus onset, and a peak magnitude at 320 ms. Topography of C2 reflected features of the MFN and an early aspect of the LPC (the P1 reprise). C4 (16% of variance) appeared broadly over parietal cortex between 530-930 ms following stimulus onset. Of note was the peaked shape of the C4 waveform peak at 760 ms. Topography was consistent with a late component of the LPC, the parietal slow wave or LPP. Components showing time course and topography consistent with the LPC (C2-C4) were retained for subsequent between-groups analyses.

Figure 6. (next page) ERP signature of self-evaluative cognition. Grand Average ERP (red = depressed, green = control), with a 200 ms baseline before stimulus onset. Black rectangle channel shown in Figure 7.



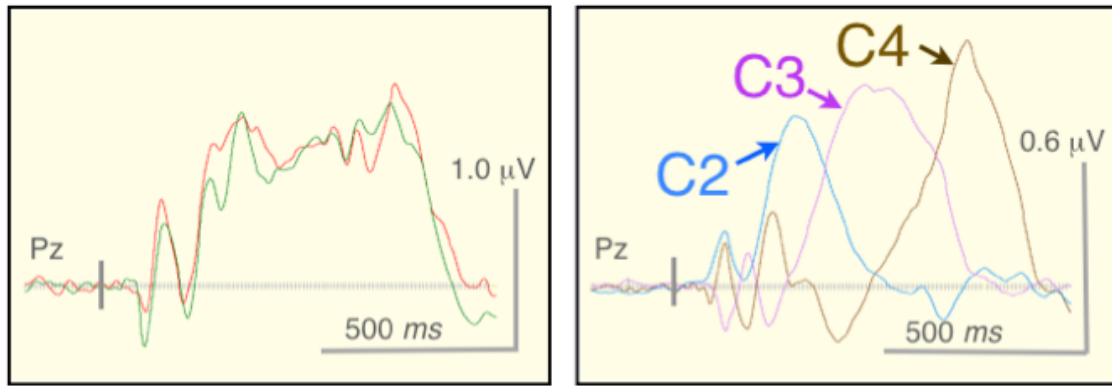


Figure 7. Decomposition of the posterior positivity in Study 1. Left: LPC at from a central parietal site (Pz) in Figure 4 shows grand average ERP (red = high BDI, green = low BDI), stimulus onset at vertical line. Right: Data decomposed into three principal components at Pz shown for the low BDI group.

Group differences in component amplitude. Relative to the low BDI group, high BDI participants showed attenuated loading on C3 over right frontal, left anterior temporal, and right parietal sites (Figure 8). In contrast, C2 was exaggerated in the high BDI group compared to low BDI participants, showing statistically significant differences over medial frontal and posterior sites. Component amplitude in C4 did not differ between control and depressed subjects. C4 was thus excluded from subsequent analyses.

Correlation of neural source activity and psychometric measures. In C3, no correlation coefficient was above the threshold for statistical significance. In C2, correlations with RBA32 (right anterior cingulate cortex) and LBA38 (left temporal pole) were above the statistical threshold. The magnitude of the RBA32 model was inversely correlated with BDI scores, $r_s(129)=-.212, p=.016$, and PNSE $r_s(129)=-.210, p=.017$. Transformed-to-normal, these relationships also appear using a parametric correlation strategy, $r(129)=-.234, p=.008$ and $r(129)=-.228, p=.009$, respectively. LBA38 was also inversely correlated with PNSE scores, $r_s(129)=-.206, p=.014$. Transformed to normal, parametric statistics maintain this association, $r(129)=-.238, p=.007$. No correlation analyses were conducted with C3 variables because between-group differences were not detected on the scalp surface.

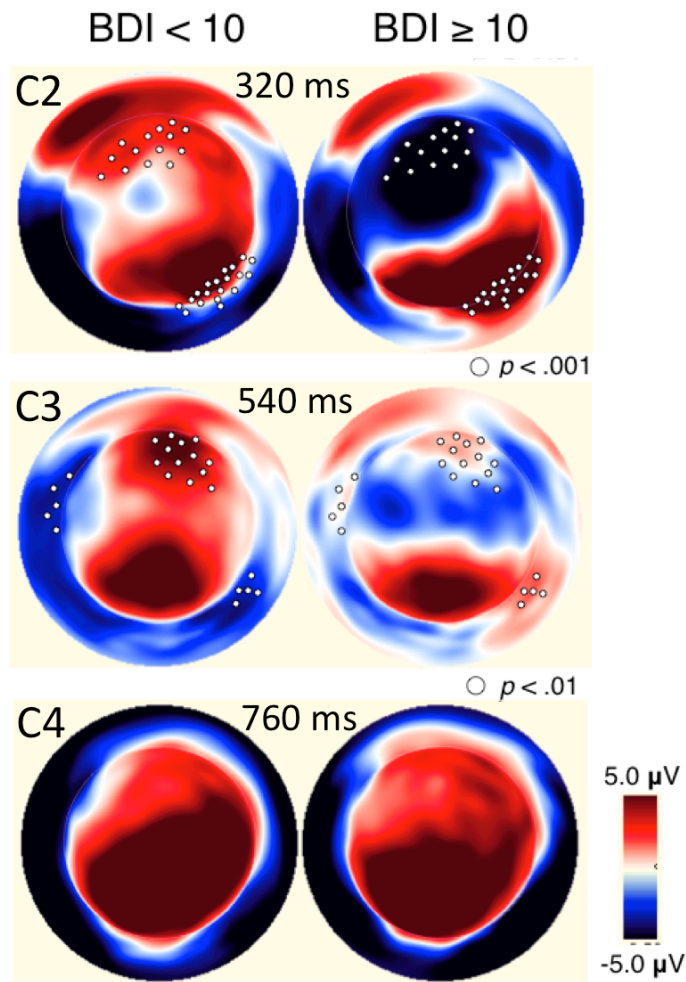


Figure 8. Topographic maps of grand average amplitude for three components. White circles identify statistically significant group mean differences. In C2, these differences reflect greater magnitude in the high BDI group. In C3, these differences reflect greater magnitude in the low BDI group.

A between-groups, means comparison was conducted, *post hoc*, on RBA32 and LBA38 in C2. Levine's test for homogeneity of variance indicated equal variance in the transformed RBA38 data, $p=.071$. Mean differences in LBA38, however, were not significant, $p=.429$. Equal variance could not be assumed in the transformed RBA32 data, $p=.014$. An independent samples t-test, adjusted for unequal variance in samples, was not significant, $p=.118$.

Neural correlates of negative self-evaluation (PNSE). In C3, RBA47 (right ventral lateral prefrontal cortex; RVLPPFC) in the high BDI group was inversely

correlated with negative self-evaluation, $r(51)=-.313$, $p=.025$. This effect was not observed in the low BDI group. Instead, RBA19 (right secondary visual cortex) in the low BDI group was inversely correlated with negative self-evaluation, $r_s(78)=-.316$, $p<.005$. This association was replicated with a parametric correlation on transformed-to-normal data, $r(78)=-.287$, $p=.011$. This effect was not observed in the high BDI group.

In C2, RBA47 (right ventral lateral prefrontal cortex; RVLPPFC) in the high BDI group was again inversely correlated with negative self-evaluation, $r_s(51)=-.339$, $p=.015$. This association was replicated with a parametric correlation on transformed-to-normal data, $r(51)=-.338$, $p=.015$. This effect was not observed in the low BDI group. Instead, LBA38 (left temporal pole) in the low BDI group was inversely correlated with negative self-evaluation, $r_s(78)=-.272$, $p=.016$. This association was replicated with a parametric correlation on transformed-to-normal data, $r(78)=-.231$, $p=.042$.

Neural correlates of depressive symptoms. In C2 RBA47 was inversely correlated with BDI, $r_s(51)=-.352$, $p=.011$ in the high BDI group. In C3, RBA38 (right temporal pole) in the high BDI group was inversely correlated with BDI scores, $r(51)=-.293$, $p=.037$. The low BDI group showed a different pattern of effects in C2: RBA47 (right ventral lateral prefrontal cortex; RVLPPFC) was positively correlated with BDI. In a *post hoc* regression analysis on the full sample ($n=129$), a quadratic model was statistically significant, $R^2=.06$, $F(2,126)=4.05$, $p=.02$, 95% CI $[-.01, .14]$ (Figure 9).

Summary of Results

What follows is a brief summary and interpretation of Study 1 results. In Chapter VII, I will provide a broader synthesis of results and discuss the implications of the findings across studies.

Behavioral results. As expected, the positive self-evaluative bias declined with increased severity of depressive symptoms. In more severe depression, scores on the symptom checklist predicted a sharper decline in positive self-evaluation. The observed inflection point in the behavioral data suggests a hypothesis that cognitive processes in severe depression might be different from the experience of transient symptoms. Based on these behavioral results, I speculated that participants with high BDI scores (i.e., depressed) may be more withdrawn. For these individuals, mood and evaluative cognition may be more constrained to internal processes (i.e., self-schema). In contrast,

participants with low BDI scores might be more engaged with the world external to themselves, which contributes noise to the self-evaluative profile on the day of testing.

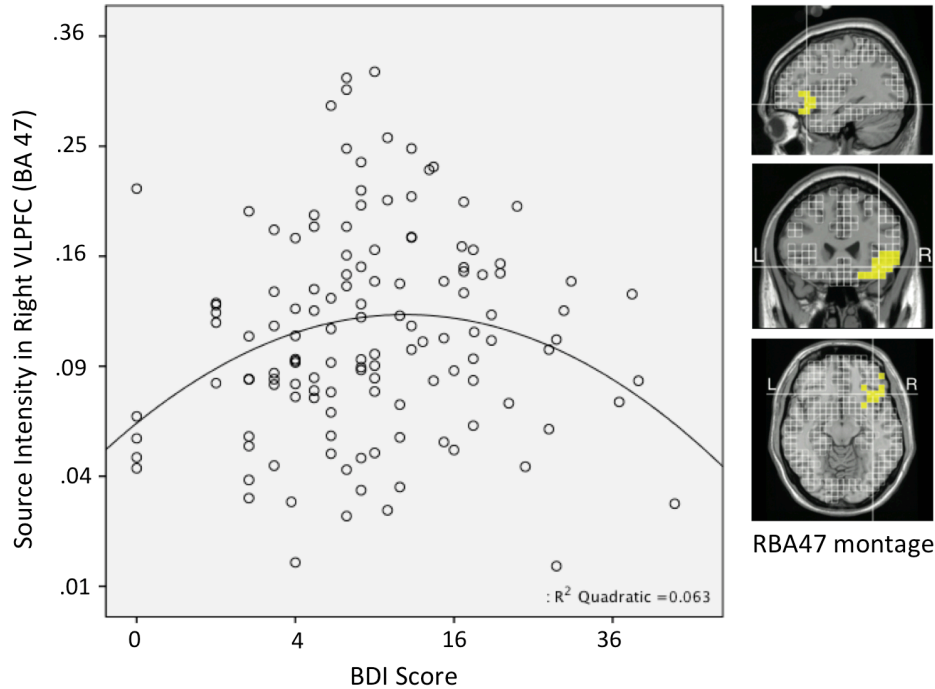


Figure 9. Right ventral lateral prefrontal cortex (RVLPFC) activity and symptom severity. x-axis: Beck Depression Inventory summary scores; y-axis: standardized measure of current density in the right BA47 (VLPFC). Right panel: BA47 dipole montage.

The dimensional specificity hypothesis, predicting patterns of mood constraint on cognition, was supported by the findings. Although both PA and NA scores predicted evaluative responses, a special association of PA with good words and NA with bad words was apparent in the effect size of the correlation statistics. Extrapolating from this finding, depression may involve more rejection of positive events, while anxiety may involve more acceptance of negative events. Future research might test the generalizability of the hypothesis beyond the context of this self-evaluation task.

C2 is exaggerated in depression: C3 is attenuated in depression. Average EEG amplitude in high BDI group (i.e., depressed) differed significantly from the low BDI group (i.e., controls). As hypothesized the component that captured variance in the P300

ERP was attenuated in depression. Extrapolating from the P300 research, C3 functionality might relate to stimulus encoding and the updating of neural schema following an emotional event. C3 topography also showed frontal cortical activity that is pathological in depression, but may only be observable with a factor analytic approach. C3 accounted for the largest amount of variance in the decision-making ERP. This may be significant because the distribution of depressive symptoms in the patient population (i.e., between-subject variance) contributed to the component structure. It appears that C3 may be relevant to dysfunctional cognition in depression.

In contrast with C3, C2 was exaggerated in the high BDI group. Although this was not a predicted finding, it may be important for understanding comorbid anxious and depressive symptoms. C2 accounted for variance in the MFN and P1r of the average ERP. Consistent with hypotheses, effects of mood-cognition interaction (i.e., between group differences) were first detected in this time window. Extrapolating from MFN research, the C3 functionality may involve high order stimulus processing relative to encoded expectations, or schema. The MFN is exaggerated in highly anxious people. Notably, participants in this experiment showed clinically significant anxiety in addition to depressive symptoms. Future research might investigate linear relationships between neural sources in C2 and symptoms of anxiety.

Consistent with previous findings, components were differentiated by wave shape; rounded in C3 and pointed in C4. However the topography of these components was not entirely consistent with the prediction. C4 showed no between-group differences and its topography was not suggestive of a ventral-anterior network. C3 showed some association with a dorsal-posterior network, however. Though speculative, the pattern of between-groups effects in C2 and C3 is more consistent with a control-systems model of frontal lobe self-regulation. This component structure reflects functions and affective biases of the dorsal-limbic system in C3, and ventral limbic system in C2.

Neural correlates of self-appraisal bias. In C2, the electrical activity in the right anterior cingulate cortex (BA32) and left temporal pole (BA38) declined with positive self-appraisal and increased symptoms severity. These findings are localized to regions of the cortex previously implicated in self-processing. However, the direction of the association is not consistent with the hypothesis.

Although EEG amplitude in C2 was exaggerated in depression, activity in the right ventral lateral prefrontal cortex (RVLPFC) was inversely correlated with symptom severity in the depressed group. In C3, trends in the depressed and control groups diverged. In the depressed group, electrical activity in the RVLPFC and right temporal pole (BA38) decreased as positive self-appraisal bias declined and symptom severity increased, as in C2. In the control group, this pattern was reversed; RVLPFC current density increased with more depressive symptoms. This quadratic association in C3 suggests an inflection point consistent with the behavioral data.

Though highly speculative, it appears that in depression there is shift away from processing environmental phenomena to an inward focus. Consistent with this hypothesis, the neural correlates of appraisal bias are also detected in the right secondary visual area the low BDI group, but not in the high BDI group.

CHAPTER V

SELF-EVALUATION AND OTHER-EVALUATION

Patterns of self-evaluation typically reflect a positivity bias (reviewed in Leary, 2007). People tend to evaluate themselves more favorably than they do another person. Self-evaluative ratings are also higher than ratings provided by another person. Individual differences in mood state and self-esteem predict the extent of the positivity bias in self-evaluation (J. D. Brown & Mankowski, 1993; Zhang et al., 2013). In clinical depression, however, the positivity bias is markedly decreased (Rimes & Watkins, 2005). In Study 1, I investigated this mood constraint on cognition as depressed and non-depressed individuals engaged in a self-evaluation task. Results of the ERP analysis showed depression-related attenuation and exaggeration at different stages of the decision-making epoch. In Study 2, the ERP signature of self-evaluation was characterized in a sample of non-depressed young adults. The goal of Study 2 was to develop a model of cortical functioning in the healthy brain that will provide a necessary contrast with results from Study 1.

Undergraduate participants were screened for subclinical levels of depression and anxiety. EEG was recorded as participants rated the extent to which a series of trait adjectives were self-descriptive, using a four-point scale. The experiment also included an other-evaluation condition, in which participants evaluated President Obama. The addition of this contrast condition addressed a limitation of the previous study; I could not conclude that effects were specifically related to self-processing. Previous applications of this contrast have shown midline effects in ERP amplitude: self- greater than other-evaluation (Esslen et al., 2008; Liu et al., 2013; Tucker, Luu, Desmond, et al., 2003).

In Study 2, I operationalized affect in two ways: as the valence of word stimuli (i.e., desirable or “good;” undesirable or “bad”), and as individual differences in trait positive and negative affect. Effects of word valence were assessed in mean differences in EEG amplitude. I also assessed the linear relationship between trait mood and EEG amplitude. I tested the hypothesis that hemispheric mood bias is asymmetrical; correlates of trait PA are right lateralized and correlates of trait NA are left lateralized. In parallel with this electrophysiological hypothesis, I sought to replicate previous findings in the

behavioral domain. I tested the hypothesis that mood constraint on self-evaluative behavior shows dimensional specificity; PA is specifically predictive of responses to good words, while NA predicts endorsement of bad words.

In Study 2, principal components analysis (PCA) was used to deconstruct the average ERP into latent factors. Results of Study 1 demonstrated that the posterior positivity, known to be influenced by mood-cognition interaction, could be decomposed into three constituent waveforms. I hypothesized that this decomposition of a posterior ERP using PCA would be replicated in Study 2, and further implicate anterior activity in affect biased cognitive processing. Results of Study 1 suggested four components, or stages, of the decision-making epoch. An aim of Study 2 was to further characterize the functional significance of each component, with focus on the interaction of emotion and cognition in neural self-regulation. I hypothesized that the component reflecting variance associated with the P300 would show effects of reference over frontal midline. I further hypothesized that effects of stimulus valence would be associated with the component that captures variance in the MFN. The statistical approach taken in this research may provide a unique window into neural network functions in self-evaluative cognition.

Methods and Materials

Participants. Forty-one (12 male) undergraduates, 18-23 years old, participated in the study for class credit. Inclusionary criteria included; right handedness, English language fluency and normal or corrected to normal vision. Exclusionary criteria included diagnosis or treatment for any of the following conditions: Traumatic Brain Injury, loss of consciousness, concussion, or dizzy spells, Epilepsy or seizures, Attention Deficit Disorder (ADD or ADHD), a neurological condition, Encephalitis, polio or other medical conditions with symptoms relating to brain function.

Procedure. Participants provided informed consent and were then fitted with a Geodesics Sensor Net (EGI, Eugene, OR). EEG was recorded as participants completed a self-evaluation task. Upon completion of the task, participants completed a series of questionnaires, including: Beck Depression Inventory (BDI; A. Beck et al., 1996), Beck Anxiety Disorder (BAI; A. T. Beck & Steer, 1990), Positive and Negative Affect Schedule-X (PANAS; D. Watson & Clark, 1999) and a debriefing questionnaire. Participants were also given a test of explicit recall of descriptor words. The experimental

session was completed in approximately two hours. The study was conducted according to protocol and practice approved by the institutional review boards of Electrical Geodesics, Inc. and the University of Oregon.

Experimental task. Descriptor words (e.g., brave) were presented, one at a time, in white on a black computer screen (Figure 10). As each word was displayed, participants indicated the extent to which the word was self-descriptive by pressing one of four buttons (1-*not at all like me*; 4-*very much like me*). In a second condition, participants instead indicated the extent to which each word described a public figure, President Barack Obama. Trials began with a central fixation point ("+") for two seconds, followed by the word stimulus for .5 seconds, and then a blank screen for an additional 1 second to allow for a response. Each of four blocks of trials lasted approximately four minutes. Condition order alternated with every participant and the direction of the scale alternated after every two participants. Participants were allowed to rest between blocks. Task stimuli and response recording was controlled by E-Prime Software, Version 1.2.1.795 (Psychology Software Pittsburgh, PA).

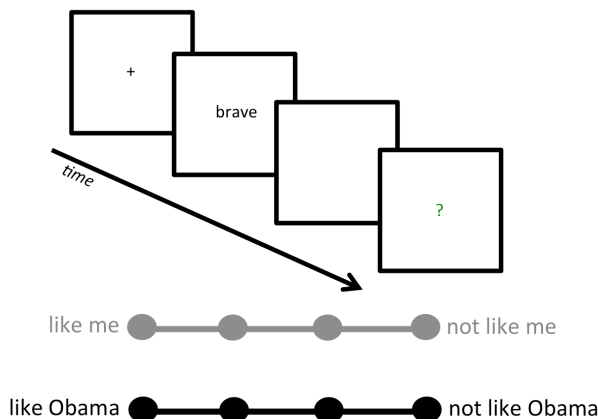


Figure 10. Schematic of stimulus presentation in Study 2. Responses in two conditions were provided on a four-point continuum (bottom).

Task development. The self-evaluation task was adapted from Tucker, et al., (2003) with a number of changes. The dichotomous response was extended to a four-point scale. Instead of providing an immediate response, participants were cued to respond 1 second after the word stimulus offset. The current study also included two conditions; self-evaluation and other-evaluation. Also, undergraduate students ($n=71$)

completed a brief survey in which they evaluated five public figures (e.g., Oprah Winfrey, Steve Jobs). Based on responding patterns and familiarity ranking, President Obama was selected for the survey.

Finally, a norming study of descriptive adjectives was conducted to select stimuli and devise categories of word stimuli that were balanced on attributes known to affect the ERP profile. Words were selected from the Oregon Self-Concept Inventory-II (OSCI-II; Tucker, Luu, Desmond, et al., 2003), PANAS-X and PANAS-C. In two separate surveys, undergraduate students rated the valence ($n=788$) and arousal ($n=207$) characteristics of each word stimulus. Response profiles were used to generate two categories of word stimuli (good and bad). 75 good words and 75 bad words were selected. The two categories of stimuli were balanced on the arousal dimension as well as word length and lexical familiarity.

EEG preprocessing. The EEG recording was made through a Net Amps 400 amplifier and Net Station software (Electrical Geodesics, Eugene, Or). The data was segmented into 1200 ms epochs, including a 200 ms pre-stimulus baseline. Non-EEG artifacts were detected and removed from the analysis using the Fully Automated Statistical Thresholding for EEG artifact Rejection (H. Nolan, R. Whelan, & R. Reilly, 2010). Nine subject files were eliminated from the analysis due to excessive non-neural signal in the recording. Trials were grouped into one of four categories, all reflecting positive appraisal. Categories reflected two level factors: reference type (self, other) and word valence (yes-good, no-bad). No-good and yes-bad trials were excluded from the analysis because there were too few to construct an ERP average. Single subject averages were re-referenced to the average reference. A grand average was also constructed for a descriptive analysis.

Statistical Analyses

Behavioral and psychometric measures. Appraisal behavior was quantified as the percent of good (or bad) words endorsed across a four-point scale. Repeated measures Analysis of Variance (rmANOVA) tested for main effects of reference (self or other), as well as the two-way interaction of valence and endorsement, and the three-way interaction of valence, endorsement and reference.

Appraisal behavior was summarized in three statistics: %-yes-to-good, %-no-to-bad, and % positive self-evaluation (PPSE), collapsed across word valence. Each of these statistics was calculated by reducing the four-point scale to a dichotomous response (*like me*; *not like me*). Trait affect was operationalized using the PA and NA scales on the PANAS survey. Correlation analyses will assess linear relationships between psychometric variables and appraisal behavior.

Principal components analysis (PCA). As in the previous chapter, statistical decomposition was conducted with the ERP Toolkit, version 2.32. A Promax rotation was used with a covariance relationship matrix and a Kaiser weighting. In this “temporal” PCA, time points are variables, analogous to items on a psychometric survey. Following decomposition into components, data were transformed back to the original scale (i.e., micro volts) by multiplying factor scores by the factor loadings for each time point (by channel by subject). The resulting waveform and scalp topography reflects the portion of the single subject ERP accounted for by each component.

Experimental contrasts in EEG data; valence and reference. Effects of experimental factors in EEG data (i.e., main effect of valence, main effect of reference, interaction of valence and reference) were characterized at each recording channel using a means contrast tool in Netstation software (EGI, Eugene, Oregon). At each channel, the average difference between two samples is compared with the standard error of that difference. The resulting statistic is assessed relative to a *t*-distribution. Results are visualized as the topographic arrangement of *t*-statistics on the head surface. The color palette reflects significance values (i.e., *p*-statistic). No statistical correction was made for multiple comparisons. The coherent spatial organization of *t*-statistics observed on the head surface is a guide to effects that survive multiple comparisons, similar to the logic of random field effects in fMRI research (Tucker et al., 1994). Following visual inspection of the *t*-test topomaps, post-hoc analysis of variance was conducted on selected channels.

Correlation analysis. Pearson’s *r* characterized linear associations between trait affect scores and selected recording channels. When Shapiro-Wilk test of normality were significant, non-parametric correlation strategies were used (Spearman’s Rho). These variables were also transformed to normal (classic logarithm) and further tested with parametric strategies. Statistical significance of the correlation coefficient was assessed

using a t -distribution. In this, the t -statistic is calculated as the ratio of the correlation coefficient to the standard error of the coefficient in the sample.

Results

Self-evaluative behavior. On average, participants engaged in positive self-evaluation on 72% (standard deviation:17) of trials and positive other-evaluation (President Obama) on 74% (22). These means were not significantly different, $p=.362$. A main effect of valence was also not significant; yes-to-good and no-to-bad trials contributed evenly to the positive appraisal bias. An interaction of reference and valence was significant, however, $F(1,41)=11.9$, $p=.001$. Simple effects showed positive evaluation of Obama involved rejection of negative words more than endorsement of positive words, $t(42)=2.1$, $p=.039$.

Positive affect (PA) and negative affect (NA) summary scores were inversely correlated, $r(41)=-.393$, $p=.011$. Positive self-evaluation inversely correlated with NA, $r(41)=-.572$, $p<.001$, but was positively correlated with PA, $r(41)=.484$, $p=.001$. Self-evaluative responses to good words correlated with both NA and PA, $r(41)=-.425$, $p=.006$; $r(41)=.423$, $p=.006$. Self-evaluative responses to bad words also correlated with both NA and PA, $r(41)=-.622$, $p<.001$; $r(41)=.465$, $p=.002$.

Positive evaluation of Obama likewise correlated inversely with NA ($r(41)=-.449$, $p=.003$) but was not predicted by PA, $p=.327$. The dimensional specificity of NA in predicting responses to negative words was supported only in the comparison of effect sizes: bad words, $r(41)=-.499$, $p<.001$; good words, $r(41)=-.364$, $p<.019$. Table 4 provides a summary of these results.

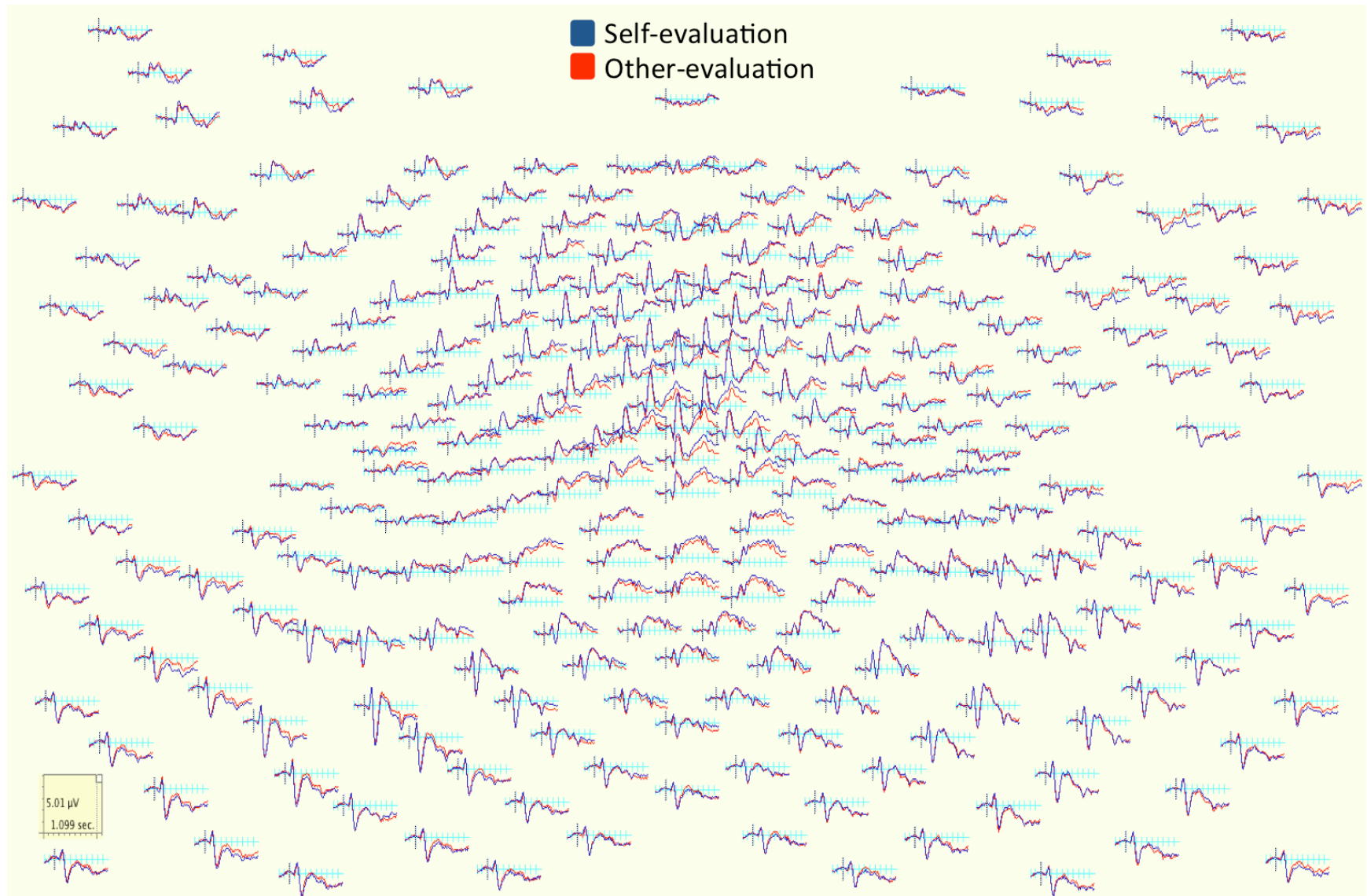
Table 4

Correlation of Trait Affect and Evaluative Behavior in Study 2

Individual Difference Measure	1	2
1. Positive Affect (PA)	--	
2. Negative Affect (NA)	-.393*	
3. Percent <i>like me</i> to good words	.423*	-.425*
4. Percent <i>not like me</i> to bad words	.465*	-.622**
5. Percent <i>like Obama</i> to good words	.140	-.364*
6. Percent <i>not like Obama</i> to bad words	.146	-.499**
$n=41$ * $p<.05$, ** $p<.001$		

Average event-related potential. The grand average ERP is shown in Figure 11. The visual P1 appeared as bilateral foci in posterior channels between 100-150 ms following stimulus onset. Peak amplitude at approximately 125 ms appeared greater in channels over the right hemisphere, as in the previous study. As the posterior positivity swept into anterior channels, the visual N1 appeared as bilateral foci in posterior channels between 150-225 ms. N1 amplitude reached a minimum at approximately 200 ms. As in the previous sample, it was left lateralized. The frontal P2 appeared between 200-270 ms and peaked at approximately 227 ms. Consistent with previous results, the frontal P2 was highly left lateralized, extending into channels over the left temple. The medial frontal negativity (MFN) appeared between 280 and 460 ms. In contrast with previous results, MFN topography appeared right lateralized in this sample. MFN amplitude reached a minimum at approximately 325 ms. A superimposed complex of positive going waves appeared over parietal cortices at 270 ms through end of the window selected for analysis (1.2 seconds). Visual inspection of the LPC waveform suggests three overlapping components. This waveform may have included the P1reprise (P1r; 270-340), the P300 (350-450ms) and the late positive potential (LPP; 470-end). The LPC appeared larger in the self-evaluation condition in contrast with other-evaluation. The MFN also appeared more robust in the self-evaluation condition, particularly in right anterior channels.

Figure 11. (next page) ERP signature of evaluative cognition in Study 2. The grand Average ERP (blue=self-evaluation, red = other-evaluation) is 1 s. long following a 200 ms baseline before stimulus onset (vertical line).



Latent factor structure of the average ERP. Based on the results of a parallel test, seven principal components were retained from the ERP data, accounting for 91% of the total variance. The topography and time course closely replicated the component structure observed in the previous analysis. A difference between the two studies, however, is seen in the extent to which individual components account for different amounts of variance. A consequence of this difference is that the components appeared in different orders across studies. To avoid confusion, I will retain the labels (C1, C2 and C3) as a reference to a component with characteristic topography and time course, while factor (F1, F2, F3) refers to the order of components in a given study. Factors 1-3 (F1-F3) accounted for three aspects of the late positive complex (LPC), each with distinct time course and topography (Figure 12). F1-F3 accounted for 36%, 20% and 18% of the total variance, respectively. Variance explained by components 4-7 (F4-F7) was: 8%, 4%, 3%, 2%.

C1 (F4) was the fourth largest component (8%). C1 appeared earliest in the decision-making epoch (140-290 ms) and was maximal at 230 ms. C1 captured variance related to the stereotyped visual ERPs: posterior N1 and P1, anterior P2. As in the average ERP, the anterior positivity extended into left ventral lateral channels. In previous analyses, only components showing time course and topography consistent with the LPC (C2-C4) were retained for subsequent analyses.

C2 (F2) was the second largest component (20% of the variance). The topography and time course of F2 closely resembled C2 in Study 1; a posterior positivity and anterior negativity between 200-550 ms that peaked at 350 ms. Topography of C2 reflected features of the MFN and an early aspect of the LPC (the P1 reprise). Of note was the asymmetrical pattern in anterior ventral lateral channels; positivity on the left and negativity on the right. In contrast with the previous analysis, the C2 waveform was more peaked than rounded.

C3 (F3) was the third largest component (18% of the variance) and it closely resembled the C3 component from the previous analysis. C3 was maximal between 400-800 ms and peaked at 600 ms. The topography of C3 reflected features of the classic P300 positivity in posterior channels. An anterior positivity observed in the previous study was notably absent.

C4 (F1) was the largest component (36% of the variance). The topography and time course of F1 closely resembled C4 in Study 1; a broad positivity over parietal cortex starting at 500 ms. C4 also captured variance related to the parietal slow wave, or late positive potential (LPP), observed in the average ERP. However, C4 differed from previous findings in that it also included an anterior positivity and the waveform had a rounded shape that appears to extend beyond the analytic time window.

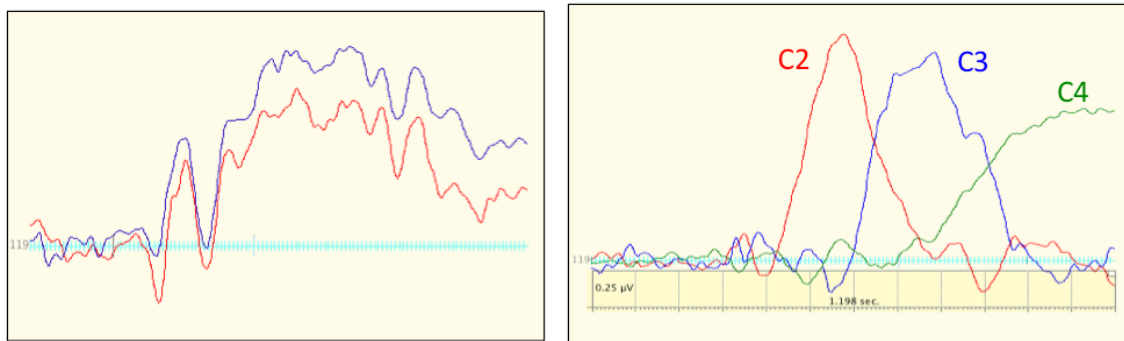


Figure 12. Decomposition of the posterior positivity in Study 2. Left: LPC at from a central parietal channel 119 (Pz). Blue= self-evaluation; red= other-evaluation. Right: Data decomposed into three principal components at channel 119 (Pz) shown for self-evaluation, only.

Effects of reference type and word valence in component amplitude. A main effect of reference type was observed across all three components (C2-C4), over central midline and in the right ventral lateral anterior channels (Figure 13). The central midline effect (self > other) fell just below a threshold for statistical significance, except for a focus over the parietal midline, $p=.06$. The right ventral lateral anterior effect (self more negative than other) was spatially coherent in C2 and neared statistical significance, $p=.057$.

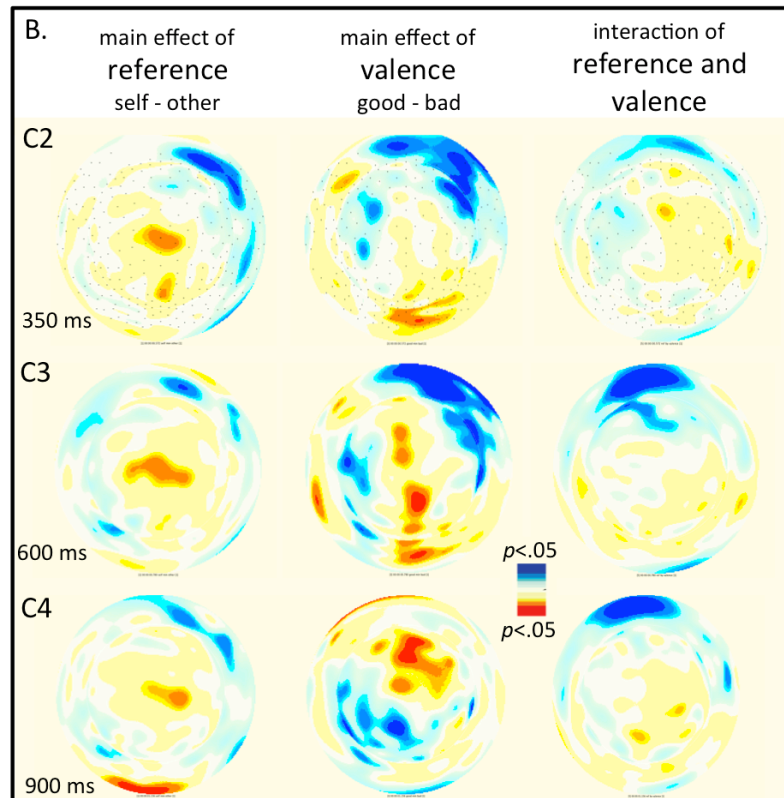
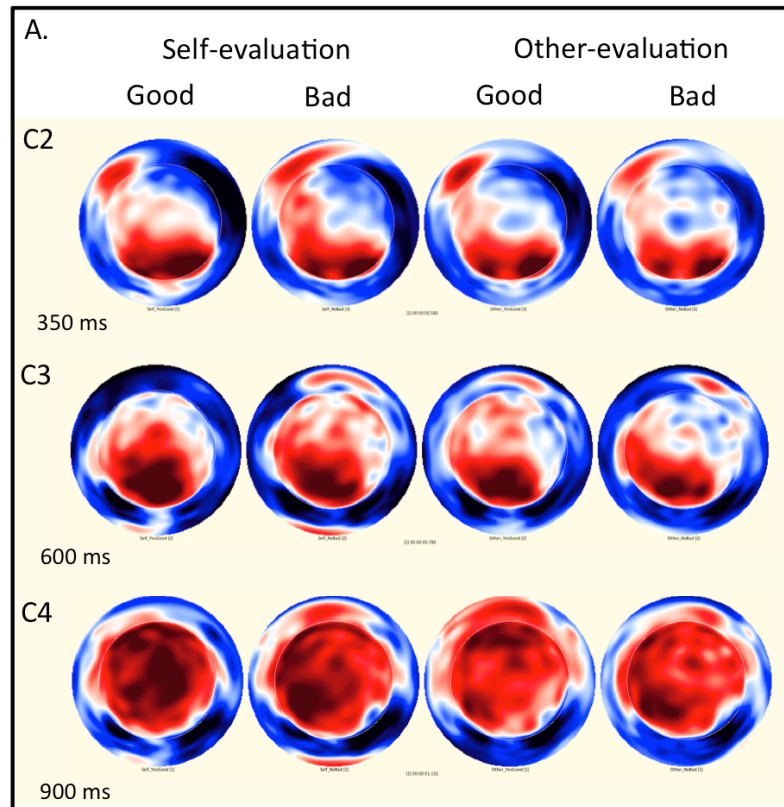
Main effects of word valence were more varied across components C2-C4. In both C3 and C2, the right ventral lateral anterior negativity was greater for good trials, $F(1,31)=12.4$, $p=.001$, $\eta^2=.286$; C3: $F(1,31)=9.7$, $p=.004$, $\eta^2=.239$. The effect was most robust and coherent in C2. Also in C2 and C3, there was a trend toward greater positivity over left superior temporal lobe and greater negativity over left ventral temporal lobe, in bad word trials. Midline and right posterior channels, in contrast, showed greater

positivity in good word trials. For C3, this effect was statistically significant over parietal midline, $F(1,31)=5.8$, $p=.022$, $\eta^2=.158$, and showed a trend over the frontal midline. In C2, this midline and right posterior effect was not statistically significant, $p=.080$. C4 effects of valence diverged from this pattern. In C4, good trials showed greater positivity in anterior channels, $F(1,31)=8.1$, $p=.008$, $\eta^2=.206$, while bad trials showed greater positivity in left posterior channels, $p=.066$.

The interaction of reference and valence was not statistically significant in C2-C4. A digital estimate of the topography beyond sensor locations suggests an interaction in ventral frontal pole. The topographical distribution of component amplitude suggests an interaction effect in the midline negativity, which appears diminished in the self-good trials. Consistent with this observation, the topography of t-statistics shows a coherent effect in midline channels. The effect is not statistically significant.

Neural correlates of trait affect. Effects of reference and word valence were asymmetric in anterior ventral lateral channels of C2 and C3. A correlation analysis investigated the relationship between trait affect and EEG amplitude. A montage of channels was selected based on the topography of statistical effects in previous analyses. Statistically significant correlations were found over the right hemisphere, but not over the left hemisphere. Right anterior effects appeared more robust for PA, than NA. PA was inversely correlated with amplitude, reflecting an increase in right ventral negativity with higher PA scores. This effect was observed in both C2 and C3 for other-evaluation, and only in C2 for self-evaluation (Figure 14).

Figure 13. (next page) Effects of reference and valence in component amplitude. A. Topographic plots of component amplitude (red=positive, blue=negative) on the scalp surface (nose pointing up). B. Topographic plot of t-statistics at each channel (p -value noted in color palette).



A post hoc repeated measures analysis of covariance (ANCOVA) was used to further assess effects in the right ventral negativity. The model included two factors, reference (self, other) and component (C2, C3), as well as two covariates, NA and PA. The between subjects effect of PA was significant, $F(1,31)=9.99$, $p<.003$, indicating that higher PA was associated with greater negativity in both C1 and C2. The between subjects effect of NA was not significant, $p=.366$. A component by PA interaction was also significant, $F(1,31)=4.6$, $p=.039$, indicating greater effects in C2 than in C3. The effect of component in self-reference observed in the correlation analysis (in C2 but not in C3) was not reflected in the results of the ANCOVA.

Table 5

Correlation of Trait Affect and Amplitude Over Ventral Lateral Prefrontal Cortex

Measure	1	2
1. Positive Affect (PA)	--	
2. Negative Affect (NA)	-.393*	
3. C2 Amplitude, <i>self</i> -evaluation	-.513**	.300
4. C3 Amplitude, <i>self</i> -evaluation	-.230	.199
5. C2 Amplitude, <i>other</i> -evaluation	-.570**	.358*
6. C3 Amplitude, <i>other</i> -evaluation	-.396*	.245
$n=32$ * $p<.05$, ** $p<.001$		

Summary of Results

Dimensional specificity in evaluative behavior. A goal of this study was to characterize the relationship between mood and self-evaluative cognition in measures of decision-making behavior and brain activity. As expected, appraisal patterns showed a positive bias. I hypothesized that trait positive and negative affect scores would show predictive specificity, such that positive affect would predict responses to good words, and negative affect would predict responses to bad words. There is only subtle evidence of dimensional specificity in these results. Both PA and NA were highly predictive of endorsement behavior, with correlation statistics in the .4 range. The inverse relationship between NA and responses to bad words (i.e., percent no-to-bad) was much stronger, with effect sizes in the -.6 range.

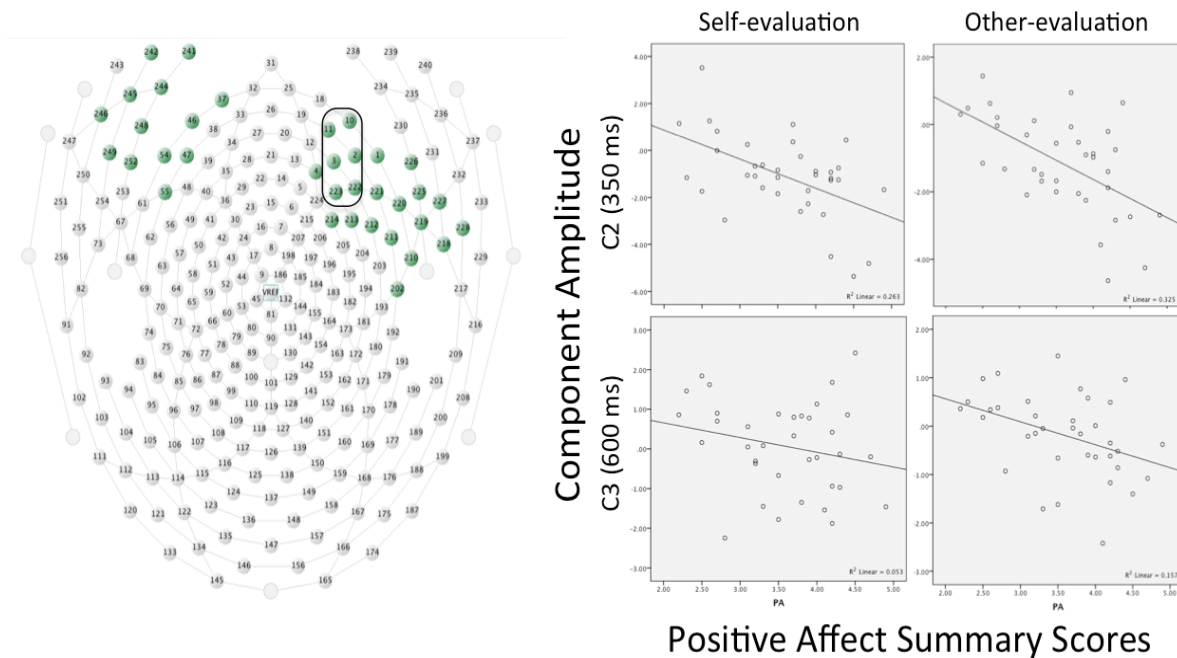


Figure 14. Correlation of PA and amplitude over ventral lateral prefrontal cortex. (Left) Schematic of EEG sensor net. green = channels included in the correlation analysis; black circle = RVLFPFC montage used in reported statistical results, including scatterplots. (Right) Scatterplots showing correlation of EEG amplitude and PA in self-evaluation (left column), other-evaluation (right column), C2 (top row), C3 (bottom row).

Evaluation of the distant other, President Obama, also provides some evidence of dimensional specificity. In this sample, NA scores predict evaluations of the President whereas PA scores do not. It may be that the construct represented by NA reflects engagement with the social environment, while PA reflects something more related to the goals of the individual. This is consistent with a two-factor structure of personality in which the NA-like factor reflects social self-regulation, while the PA-like factor reflects dynamism in the individual (Saucier et al., 2014). On average, participants did not rate themselves more favorably than the President. This is contrary to research in this area that typically shows higher self-rating compared to evaluation of a close or distant other (Leary, 2007). Notably, positive appraisal of the President was populated by no-to-bad trials, while no-to-bad and yes-to-good trials were evenly represented in positive self-

evaluation. It appears that these undergraduate participants are influenced more by NA (i.e., social self-regulation) than PA, when evaluating the President at the height of his popularity.

Components of the decision-making ERP. As hypothesized, the parietal positive complex was decomposed into three separate factors. The onset of each is coincident with the onset observed in the ERP. In order of onset: the P1r (C2), P300 (C3) and LPP (C4). ERP research has variously associated features of the posterior complex with decision-making behaviors and shown effects of trait affect as well as stimulus valence (G. Hajcak et al., 2010). An obstacle to interpretation of these effects is that the average ERP waveform represents a superposition of multiple processes over posterior cortices. The current results demonstrate that the posterior complex is reliably deconstructed into three components. In addition, the component topography shows anterior activity that is highly correlated with each posterior component. Because these latent factors represent non-overlapping electrical activity and account for the entire scalp surface, results are more amenable to interpretation relative to the mechanics of electrical brain function.

A subsequent goal of this research was to begin to characterize the functional nature of these components. I used experimental contrasts to identify affective and cognitive attributes in each component. I began with a hypothesis from the previous study that C3 (P300) would show effects of reference (self, other), and that C2 (P1r-MFN) would show effects of stimulus valence. Results, however, suggest a more complicated picture.

The self in component structure and amplitude. To understand the neural mechanics of self-evaluation in particular, I developed a contrast condition that was identical in every way but one: the person under evaluation. In ERP research the difference between self-evaluation and the evaluation of a close friend is very subtle. There is also evidence from brain imaging that as affiliation decreases, the distinction between self- and other-reference increases. I sought to exaggerate the difference between self and other by selecting a public figure that is known by most participants, but not closely affiliated with any of them. Despite this conceptual difference, the main effect of the contrast in component amplitude was subtle. Consistent with previous results

(Tucker, Luu, Desmond, et al., 2003), it was difficult to distinguish self from other reference in the electrophysiological signature of an evaluative decision.

Though below statistical threshold, mean differences between self- and other-reference (self>other) spanned the dorsal midline, with effects growing stronger over parietal cortex. This may reflect additional processing in the posterior cingulate cortex, and central midline structure (CMS). Self- was also more robust than other-evaluation over the right ventral lateral prefrontal cortex (rVLPFC). The effect is present in all three components, but statistically significant in C2 (P1r-MFN). Indeed, this is strongest observed effect of the contrast. In summary, effects of reference did not differentiate components. However, the midline trend (self>other) contextualizes the current effect among previous reports. In addition, the rVLPFC is implicated (self- more negative than other-reference), particularly in C2. It may be that the rVLPFC plays an important role in evaluative decision-making.

Affect-cognition interaction and a control-systems hypothesis. Results show effects of word valence in the electrophysiological signal. The pattern of results also differentiated the three components. In the latest component, C4 (LPP), good words evoked a larger positivity over the frontal midline, and bad words evoked a larger positivity in in left posterior channels. This interaction of valence and topography is particularly interesting given that C3 begins around 600 ms, likely after the evaluative decision has been made. Closer inspection of the component topography suggests several effects that might contribute to the posterior difference: lack of left posterior focality for other-good and the shape of the self-good focus over central midline. Although the interaction of valence and reference was not significant over left posterior channels, this observation is consistent with previous reports of greater LPP in self-reference and in response to positive stimuli.

The time course of C2, followed by C3, is more consistent with initial decision-making processes. In both components, bad is more positive than good over left temporal lobe. This may reflect additional processing of negative stimuli in language systems of the lateral left hemisphere. In C3 (P300) good is more positive in posterior midline channels. This is consistent with numerous reports that show P300 larger for positive stimuli. The component topography, however, further implicates central and anterior

midline channels, suggesting that valence processing in CMS structures is uniquely captured in C3. In both C3 and C2, the negativity over right ventral lateral prefrontal cortex (RVLPFC) is once again implicated (good more negative than bad). As in the contrast of self and other, the RVLPFC is particularly robust in C2 (P1r-MFN). Taken together, C2, the earliest of the three components, appears to capture variance related mood-cognition interaction.

Differentiating frontal control systems in the component structure. A tertiary goal for this research is to elaborate on a control-systems model of frontal lobe function (and dysfunction). Statistical decomposition of the average ERP uses correlated neural activity to define a latent factor structure. The functional significance of that factor structure is unknown. According to the control-systems model, it is the dynamic reciprocity of limbic-cortical, dorsal-limbic and ventral-limbic systems, right and left hemispheres that gives rise to cognition (Tucker & Luu, 2012; Waters & Tucker, 2013a). Of note in the C2 and C3 topography is an asymmetric pattern of electrical potentials in lateral anterior channels. Consistent with a dimensional model of affect motivated control systems in the frontal lobe, I hypothesized that activity in the right hemisphere is biased by PA; while activity in the left is biased by NA. To test this hypothesis, I conducted a post-hoc analysis on the asymmetric activity in C2 and C3. PA was strongly and significantly correlated with amplitude in right but not left anterior ventral lateral channels. This effect appeared in both self- and other-evaluation and was more robust in C2. This result is consistent with the hypothesized model and further implicates RVLPFC in the affective processing during self-evaluation.

Further association of the latent factor structure with the hypothesized model is highly speculative. Features of C2 are more consistent with ventral limbic functioning. The experimental effects show activity related to stimulus processing in C2 and frontal effects are lateralized over ventral cortex. C2 captures variance related to the MFN and P1r, two average ERP waveforms that are associated with re-entrant, or higher order cognitive processing of a stimulus or event (P. Luu et al., 2000). In contrast, the topography of C3 and C4 is more dorsal in nature. C4, in particular, shows effects of stimulus processing over broad swaths of anterior and posterior dorsal cortex. C3 is associated with both midline and ventral lateral effects. Though not consistent with the

basic outline of a control-systems model, this association of C3 with central midline structures, as well as the classic P300 ERP, is suggestive of the central midline network implicated in self-processing (Qin & Northoff, 2011).

CHAPTER VI

SELF-EVALUATION AND SEMANTIC CATEGORIZATION

The third study in this series was designed to replicate and build on the previous results. Young adult undergraduates engaged in a self-evaluation task while their EEG was recorded. In addition to the self-evaluation task, participants completed a semantic categorization task. This contrast condition was identical to the self-evaluation task in every way but instruction. In the categorization task, participants rated the extent to which each word described an emotion or a personality trait, presented as a bipolar scale. The contrast was used to identify self-specific processing in the decision-making ERP.

As in Study 2, this research was aimed at establishing a model of neurotypical brain function during self-evaluative cognition. Results may serve as a baseline contrast for research with clinical samples. A primary analytic goal of Study 3 was to assess the replicability of the component structure, time course and topography. Through replication of exploratory findings, Study 3 also contributed to the central goal of this research; to assess a network model of affective self-regulation in self-evaluative decision-making.

Hypotheses reflected these central goals, as well as the assumptions of an affective-arousal control-systems model. I hypothesized that positive-self appraisal bias would be predicted by measures of mood state. Specifically, there would be dimensional specificity in the predictive relationship such that PA will be associated with endorsement of good words, and NA with bad words. Extended to neural measures, I hypothesized that there would be dimensional specificity in hemispheric bias. In particular, I predicted that trait PA would correlate with activity over the right ventral lateral prefrontal cortex (RVLPFC), as seen in Study 2. Seeking to replicate the findings of Study 2, I predicted that statistical decomposition of the average ERP would separate the posterior positivity into three components. A goal of this investigation was to clarify the neural mechanisms of self-appraisal behavior, with particular focus on those that involve affective processes. This goal was approached with the characterization of mood and valence effects in component amplitude. Results were interpreted relative to an affective-arousal model of neural self-regulation.

Methods and Materials

Participants. Forty-three (14 male) undergraduates, 18-24 years old, participated in the study for class credit. Inclusionary criteria included; right handedness, English language fluency and normal or corrected to normal vision. Exclusionary criteria included diagnosis or treatment for any of the following conditions: Traumatic Brain Injury, loss of consciousness, concussion, or dizzy spells, Epilepsy or seizures, Attention Deficit Disorder (ADD or ADHD), a neurological condition, Encephalitis, polio or other medical conditions with symptoms relating to brain function.

Procedure. Participants provided informed consent and were then fitted with a Geodesics Sensor Net (EGI, Eugene, OR). EEG was recorded as participants completed a self-evaluation task. Upon completion of the task, participants completed a series of questionnaires, including: Beck Depression Inventory (BDI; A. Beck et al., 1996), Beck Anxiety Disorder (BAI; A. T. Beck & Steer, 1990), Positive and Negative Affect Schedule-X (PANAS; D. Watson & Clark, 1999) and a debriefing questionnaire. The experimental session was completed in approximately two hours. The study was conducted according to protocol and practice approved by the institutional review boards of Electrical Geodesics, Inc. and the University of Oregon.

Experimental task. Descriptor words (e.g., brave) were presented, one at a time, in white on a black computer screen (Figure 15). As each word was displayed, participants indicated the extent to which the word was self-descriptive by pressing one of four buttons (1-*not at all like me*; 4-*very much like me*). In a second condition, participants decided to what extent each word described a personality trait or a mood state (1 - mood state, 4 - personality trait). Trials began with a central fixation point ("+") for 2 seconds, followed by the word stimulus for .5 seconds, and then a blank screen for an additional one second to allow for a response. Condition order alternated with every participant and the direction of the scale alternated after every two participants. Each of four blocks of trials lasted approximately four minutes. Participants were allowed to rest between blocks.

This self-evaluation task was adapted from Tucker, et al. (2003) and Study 2, in this series. Word stimuli were identical to those used in Study 2. Good word and bad word groups of 75 words each were balanced on several attributes; arousal, magnitude of

valence, word length and lexical familiarity. Norming procedures are described in Chapter V. Task stimuli and response recording was controlled by E-Prime Software, Version 1.2.1.795 (Psychology Software Pittsburgh, PA).

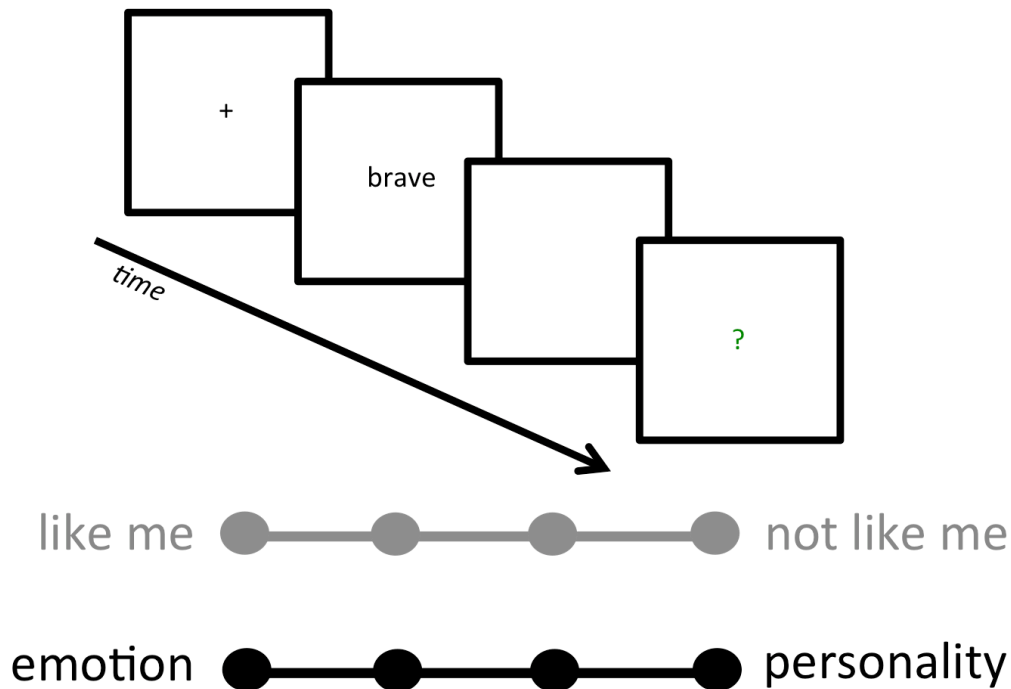


Figure 15. Schematic of stimulus presentation in Study 3. Responses in two conditions were provided on a four-point continuum (bottom).

EEG preprocessing. The EEG recording was made through a Net Amps 400 amplifier and Net Station software (Electrical Geodesics, Eugene, Or). The data was segmented into 1200 ms epoch; including a 200 ms pre-stimulus baseline. Non-EEG artifacts were detected and removed from the analysis using the Fully Automated Statistical Thresholding for EEG artifact Rejection (H Nolan et al., 2010). Seven subject files were eliminated from the analysis due to excessive non-neural signal in the recording. There were two experimental factors in the analysis: task and word valence. Trials were grouped into one of four categories: (self-evaluation, good; self-evaluation, bad; categorization, good; categorization, bad). Single subject averages were re-

referenced to the average reference. A grand average was also constructed for a descriptive analysis.

Statistical Analyses

Behavioral and psychometric measures. Self-evaluative behavior was quantified as the percent of good (or bad) words endorsed on the four-point scale. This scale was collapsed to a dichotomous variable for statistical analysis. Repeated measures Analysis of Variance (rmANOVA) tested for main effects of valence, as well as the two-way interaction of valence and endorsement. In a separate analysis, effects of word valence on semantic categorization behavior was assessed using rmANOVA.

Self-evaluative behavior was summarized in three statistics: %-yes-to-good, %-no-to-bad, and an overall summary statistic, % positive self-evaluation (PPSE). Trait affect was operationalized using the PA and NA scales on the PANAS survey. Correlation analyses assessed linear relationships between psychometric variables and appraisal behavior.

Principal components analysis (PCA). As in the previous chapter, statistical decomposition was conducted with the ERP Toolkit, version 2.32 (Dien, 2010). A Promax rotation was used with a covariance relationship matrix and a Kaiser weighting. In this “temporal” PCA, time points are variables, analogous to items on a psychometric survey. Following decomposition into components, data were transformed back to the original scale (i.e., micro volts) by multiplying factor scores by the factor loadings for each time point (by channel by subject). The resulting waveform and scalp topography reflects the portion of the single subject ERP accounted for by each component.

Experimental contrasts in EEG data; word valence and task type. Effects of experimental factors in EEG data (i.e., main effect of word valence, main effect of task, interaction of valence and task) were characterized at each recording channel using a means contrast tool in Netstation software (EGI, Eugene, Oregon). At each channel, the average difference between two samples is compared with the standard error of that difference. The resulting statistic is assessed relative to a *t*-distribution. Results are visualized as the topographic arrangement of *t*-statistics on the head surface. The color palette reflects significance values (i.e., *p*-statistic). No statistical correction was made for multiple comparisons. The coherent spatial organization of *t*-statistics observed on the

head surface is a guide to effects that survive multiple comparisons, similar to the logic of random field effects in fMRI research (Tucker et al., 1994). Following visual inspection of the t-test topomaps, post-hoc analysis of variance was conducted on selected channels.

Directed correlation analysis. Pearson's r characterized linear associations between trait affect scores and selected recording channels over ventral lateral prefrontal cortex (Figure 16). When Shapiro-Wilk tests of normality were significant, non-parametric correlation strategies were used (i.e., Spearman's Rho). These variables were also transformed to normal (classic logarithm or square root) and further tested with parametric strategies. Statistical significance of the correlation coefficient was assessed using a t -distribution. In this, the t -statistic is calculated as the ratio of the correlation coefficient to the standard error of the coefficient in the sample.

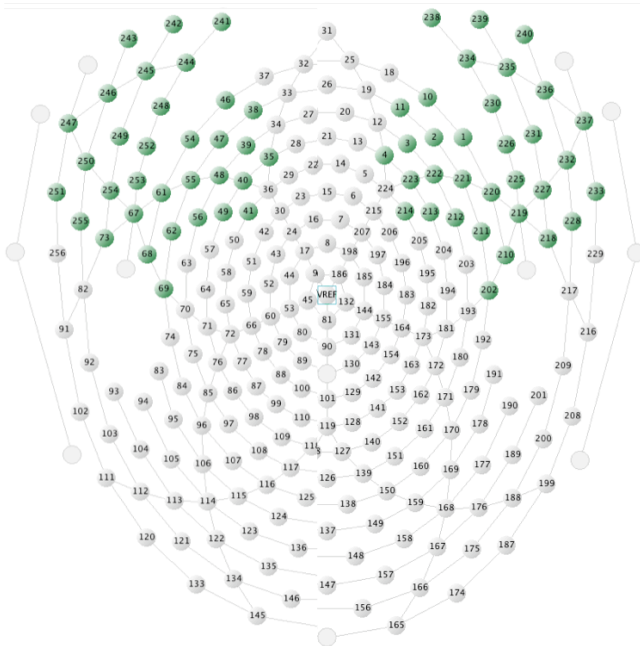


Figure 16. Channels selected for the correlation analysis.

Results

Self-evaluative behavior. On average, participants engaged in positive self-evaluation on 75% of trials. A main effect of valence was not significant; yes-to-good and no-to-bad trials contributed evenly to the positive appraisal bias. In the semantic

categorization task, participants more often classified words as a personality descriptor (57%) than a mood descriptor, $F(1,41)=30.1, p<.001$. The interaction of word valence and category was also significant as participants tended to classify bad words as mood states and good words as personality traits, $F(1,41)=269.8, p<.001$.

The median positive affect (PA) summary score was 36 (SD 5.5), and the median negative affect (NA) summary score was 20 (SD 4.3). PA and NA scores were not correlated with each other ($p=.172$). The median BDI score was 8, (SD=6.7) and the median BAI score was 7 (SD=7). BDI and BAI were highly correlated, $r_s(41)=.562, p<.001$. NA was positively correlated with BDI, $r_s(41)=.363, p=.017$, and BAI, $r_s(41)=.343, p=.025$. An inverse correlation of PA and BDI neared significance, $r_s(41)=-.298, p=.053$.

There was dimensional specificity in the prediction of endorsement behavior (Table 6). PA was correlated with positive self-evaluation $r_s(41)=.514, p=.001$ overall, and with the endorsement of good words, $r_s(41)=.325, p=.036$, and rejection of bad words, $r_s(41)=.428, p=.001$. NA was not correlated with self-evaluative behavior statistics. Neither BDI or BAI predicted self appraisal in general, but they did predict responses to bad words: BDI: $r_s(41)=-.447, p=.003$; BAI: $r_s(41)=-.359, p=.018$.

Table 6

Correlation of Trait Affect and Self-Evaluative Behavior in Study 3

Individual Difference Measure	1	2	3
1. Positive Affect (PA)	--		
2. Negative Affect (NA)	-.135	--	
3. Percent <i>yes</i> to good words	.325*	.051	--
4. Percent <i>no</i> to bad words	.482**	-.181	.410**

$n=41$ * $p<.05$, ** $p<.001$

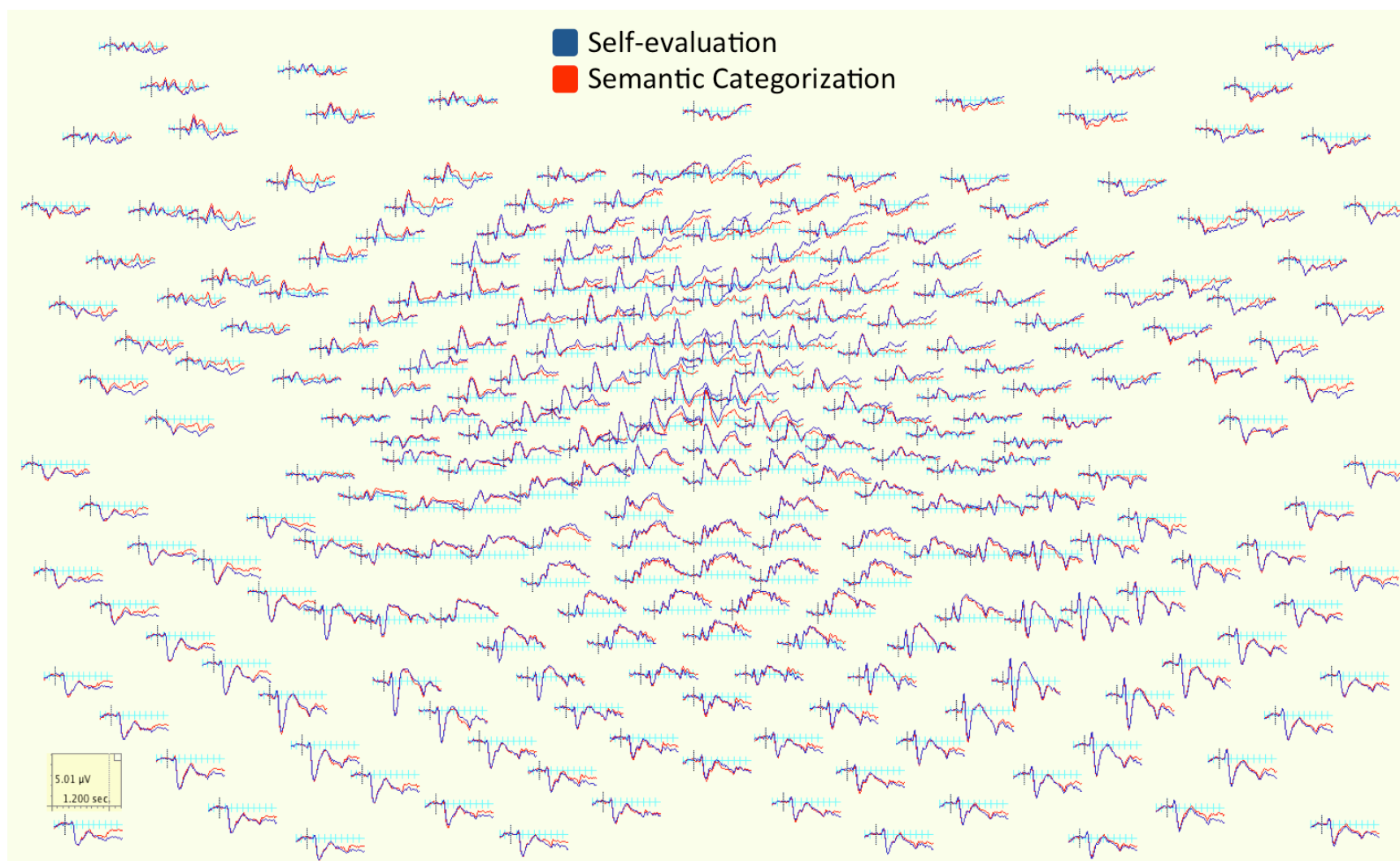
Average event-related potential. Figure 17 shows the grand average ERP for self-evaluation and the categorization task. The visual P1 appeared as bilateral foci in posterior channels between 100-140 ms following stimulus onset. The P1 was strongly

right lateralized in this sample; peak amplitude at approximately 120 ms was minimal in left posterior channels. As the posterior positivity swept into anterior channels, the visual N1 appeared as bilateral foci in posterior channels between 150-300 ms. N1 amplitude reached a minimum at approximately 200 ms. As in the previous sample, N1 amplitude appeared greater on the left. The frontal P2 appeared between 195-240 ms and peaked at 215 ms. Consistent with previous results, the frontal P2 was highly left lateralized, extending into channels over the left temple. The medial frontal negativity (MFN) appeared between 310 and 440 ms, and reached a minimum at approximately 400 ms. A superimposed complex of positive going waves appeared over parietal cortices from 260-900ms. Visual inspection of the LPC waveform suggests three overlapping components. This waveform may have included the bilateral P1reprise (P1r; 270-445), the central P300 (450-550ms) and late positive potential or parietal slow wave (LPP; 550-900). The P1r appeared to transition into the P300 earlier in self-evaluation. In semantic categorization, MFN also appeared more negative in the self-evaluation condition, particularly in right anterior channels. In the second half of the epoch, a left lateralized, anterior positivity appeared greater for self-evaluation than semantic categorization.

Latent factor structure of the average ERP. Based on previous findings, a seven-factor structure was retained from the ERP data, accounting for 92% of the total variance. Notably, results of a parallel test suggested that a six-factor structure would account for a similar amount of variance. The topography and time course closely replicated the component structure observed in the previous analysis (Figure 19).

C1 was the fourth largest component (9%). C1 was the earliest component (380-480 ms) and was maximal at 430 ms. C1 captured variance related to the stereotyped visual ERPs: posterior N1 and P1, anterior P2. As in the average ERP, the anterior positivity extended into left ventral lateral channels.

Figure 17. (next page) ERP signature of evaluative cognition in Study 3. The grand Average ERP (blue=self-evaluation, red = other-evaluation) is 1 s long following a 200 ms baseline before stimulus onset (vertical line).



C2 was the second largest component (27% of the variance), as in the previous study. The C2 waveform was maximal between 400ms-850ms and peaked at 400 ms. C2 topography reflected the MFN and an early aspect of the LPC (the P1 reprise) of the average ERP. Of note was the asymmetrical pattern in anterior ventral lateral channels; positivity on the left and negativity on the right.

C3 was the third largest component (13% of the variance). The C3 waveform was maximal between 400-850 ms and had a rounded shape with a peaked feature at 680 ms. The topography of C3 reflected features of the classic P300 positivity in posterior channels.

C4 was the largest component (31% of the variance), as in the previous study. Starting at 450 ms, the C3 waveform was rounded in shape and extended beyond the analytic window. This component captured variance related to the parietal slow wave, or late positive potential (LPP), observed in the average ERP. C3 topography was a broad, right lateralized positivity in posterior channels and a central positivity in anterior channels.

C5-C7 accounted for 5%, 4% and 3% of the variance, respectively.

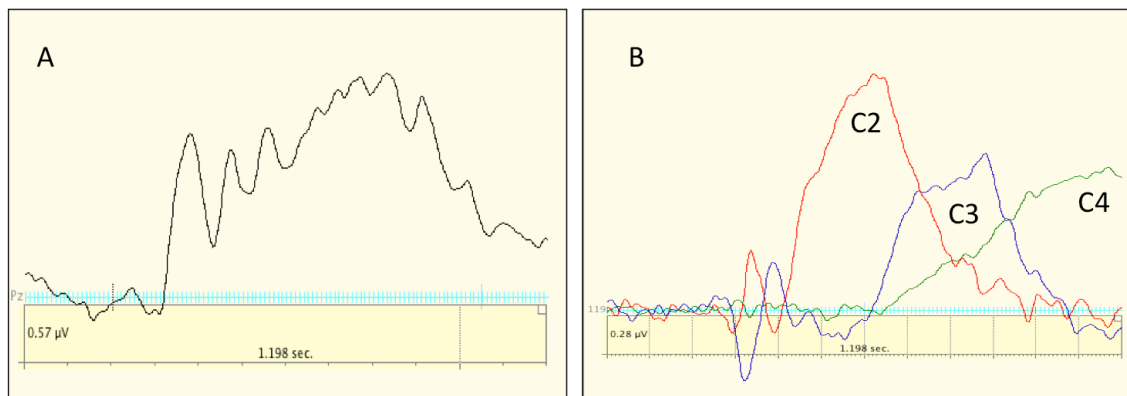


Figure 18. Decomposition of the posterior positivity in Study 3. Left: LPC from a central parietal channel 119 (Pz). Right: Data decomposed into three principal components at channel 119 (Pz). Both panels reflect self-evaluation, only.

Effects of task type and word valence in component amplitude. Figure 20 provides topographic maps of component amplitude and statistical testing. There was a main effect in C4, where self-evaluation was more positive in anterior channels, $F(1,35)=21.65, p<.001, \eta^2=.382$. Though not statistically significant self-evaluation was

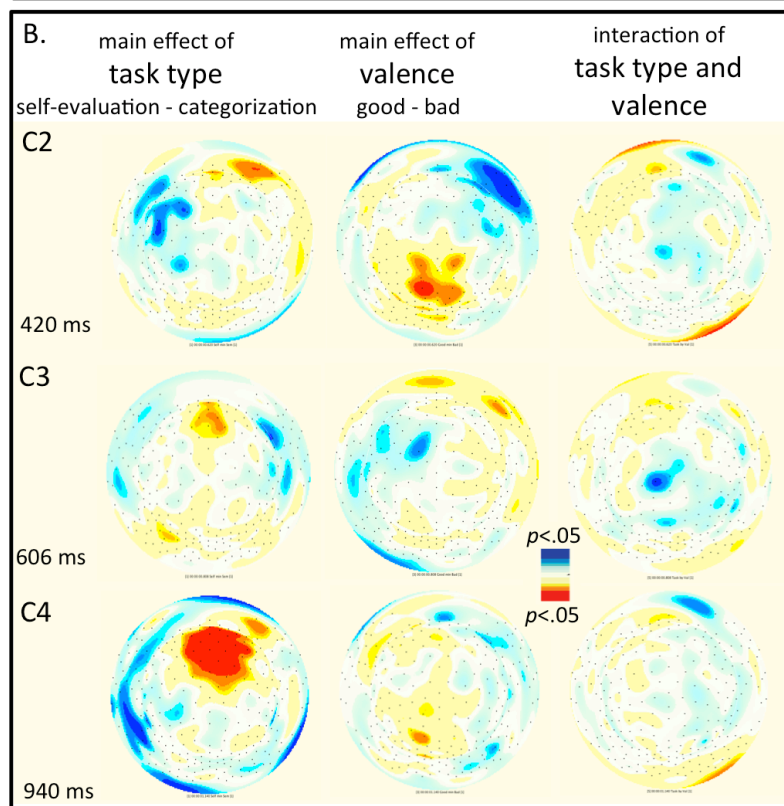
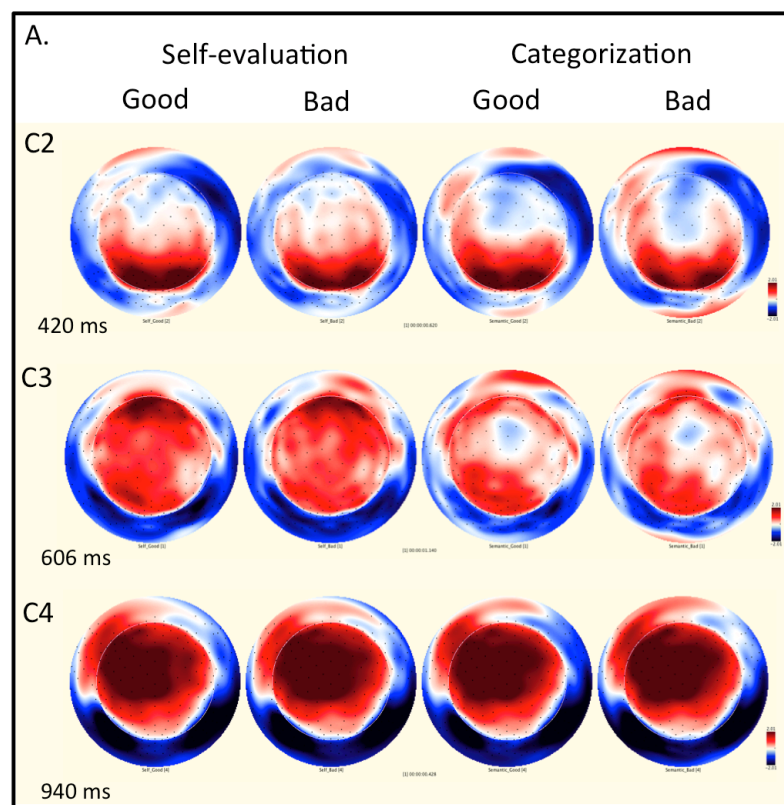
more robust than the semantic task in the left lateral negativity over temporal cortex, across all components. This effect is most robust in C2, where voltage in anterior lateral channels appears asymmetric. The distribution of t -statistics in C2, was consistent with the absence of a left frontal positivity in self-evaluation, and an attenuated right anterior ventral negativity in the semantic tasks. In C3, the statistical distribution, though below threshold for significance, suggests self-evaluation more positive in anterior midline channels, and more negative in anterior ventral channels, particularly on the right.

C2 showed statistically significant mean differences between good and bad trials in right ventral lateral channels, $F(1,35)=13.12$, $p=.001$, $\eta^2=.272$. Good word trials were more negative than bad word trials. Good word trials were more positive than bad word trials in central and right posterior channels, $F(1,35)=17.9$, $p<.001$, $\eta^2=.338$. The distribution of t -statistics in C3 suggested asymmetric effects in the dorsal positivity; good greater than bad on the right and bad greater than good on the left. This pattern was reversed in C4.

No interaction effect was statistically significant in C2-C4. Although the t -distribution suggests that the medial negativity is greater in the semantic task, there is more evidence of an interaction effect in C2 average topomaps; the medial negativity is larger for good words in the self-evaluation task, and for bad words in the semantic task.

Neural correlates of trait affect. No statistically significant correlation of trait affect (NA and PA) and component amplitude was found in C2 or C3.

Figure 19. (next page) Effects of task type and valence in component amplitude. A. Topographic plots of component amplitude (red=positive, blue=negative) on the scalp surface (nose pointing up). B. Topographic plot of t -statistics at each channel (p -value noted in color palette).



Summary of Results

Measuring frontal lobe control systems. In time course and topography, the factor structure in Study 3 replicated findings in Study 2. The posterior positive complex was again decomposed into three separate components, with anterior topography comparable to the previous study. The C4 waveform replicated the time course observed in Study 2. This further supports the hypothesis that the shifted latency in C4 may be related to response time, which was delayed in Study 2 and Study 3, but immediate in Study 1. C2 was broader in Study 3 and the midline negativity was attenuated relative to previous findings.

I then looked at the effects of two experimental variables (i.e., task type and word valence) in component amplitude. Effects of valence in word stimuli was primarily associated with C2 (starting at 200 ms). For both categorization and self-evaluation, good words evoked greater positivity in posterior channels. The most robust effect of word valence was over right ventral lateral frontal cortex in C2; replicating findings from the previous study. It appears that activity in this area represents an anterior correlate of the posterior P1r; a negativity that, like the P300 positivity, is more robust when the brain is processing pleasant or positive stimuli.

Though not statistically significant, there was an earlier effect of task type, from 200 ms to 600 ms in C2. This effect was localized to an anterior ventral positivity that appeared over the left hemisphere, and was more coherent in the semantic condition. The left hemisphere is preferentially involved in language processing; and the acquisition of semantic meaning might occur within this time window (Neville & Bavelier, 1998). In the left temporal pole, the language path is connected to ventral limbic structures, such as the amygdala. It may be that basic language processing in the categorization task recruited more ventral limbic engagement than in the self-evaluation task. Taken together, effects of valence and task type in C2 suggest asymmetric engagement of the frontal lobe; language-related differences on the left and emotion processing on the right.

The most robust finding was a main effect of task type in C4. Self-evaluation was greater than categorization in the second half of the epoch. This effect is most consistent with previous reports (Esslen et al., 2008; Liu et al., 2013). It is notable that there was no

effect of word valence in C4, suggesting that affective attributes of the stimulus were irrelevant to the processes represented by this factor.

Neural correlates of trait mood in the decision-making epoch. In a second analysis, I identified neural correlates of trait mood. In contrast with results of the previous study, there was no significant correlation of trait affect scores and component amplitude in selected channels. This is surprising given the robust nature of the finding in the previous sample. It is notable that NA and PA scores were not correlated in Study 3. Although the constructs are theoretically orthogonal, PA and NA scores are often correlated. Also notable was the failure to replicate the dimensional specificity hypothesis; that PA would predict responses to good words and NA would predict responses to bad words. In Study 3, PA predicted endorsement patterns, while NA did not. It is unclear why these patterns of correlation differ between Study 2 and Study 3.

CHAPTER VII

SUMMARY AND SYNTHESIS

This research is an investigation of how affective and cognitive processes interact in the frontal lobe during self-evaluative decision-making. Although mood-cognition interaction could be studied in a variety of experimental contexts, self-evaluation is the focus of this research for several reasons. First, there is an apparent association of mood state and evaluation bias in behavioral measures. This provided a pattern of variance that could be subsequently identified in neural measures. Second, self-evaluation bias is used to describe core symptoms of mood and anxiety disorders. Some treatments target self-evaluative behavior directly, and self-reported emotional experience is commonly used to measure treatment efficacy. This alignment with psychometrics was central to the research strategy; it provided a unique opportunity to convolve ERP analysis with psychometric methods. Finally, emotional decision-making evokes functions of frontal lobe, self-regulatory systems. Whether implicit or explicit, self-appraisal is inherent to on-going, social self-regulation. Characterization of frontal lobe mechanisms in this context provides insight into the neural basis of adaptive and dysfunctional social behavior.

In this chapter, I provide a synopsis and tentative interpretation of the findings. I begin with a brief review of the control-systems framework. This model posits dimensional affective-arousal bias in neural self-regulation. I explore how psychological phenomena, including the positivity bias in self-evaluative cognition, are an emergent property of these reciprocal control systems. In this, cognition is viewed as a process. A cognitive act involves the projection of a predictive model (i.e., schema) and experience dependent updating of that model. These stages of processing are also represented in components of the decision-making ERP.

I then explore classical theories of depression (e.g., learned helplessness) through the lens of neural self-regulation. I extend this discussion to the brain basis of psychotherapeutic, psychopharmacological and neural stimulation treatments for depression. I suggest that the loss of positivity bias in depression may disrupt the process of cognition by limiting the extent to which learned schema are updated through

interaction with the environment. Co-morbid anxiety, I argue, may be an adaptive compensation for loss of positive arousal that effectively drives attention outward.

In this research, I implemented two analytic strategies to elucidate network dynamics in self-evaluative cognition. Both convolve psychometric methods with ERP analysis. I first review rationale and findings of the neuropsychometric approach. These findings guided the subsequent investigation in which I used factor analysis to deconstruct the decision-making ERP. As hypothesized, the factor analysis reduced the posterior positivity into three components. This component structure was replicated in three separate samples.

A central goal of this research was to characterize effects of depression in the component structure. I review the results of Study 1, which involved depressed and non-depressed adults. Results showed between group effects in component 2 (C2) and component 3 (C3). I return briefly to non-neural measures and address the hypothesis that trait mood (PA and NA) each predict different aspects of self-evaluative behavior. Results are mixed but suggest some future directions for research in this area. A related hypothesis was directed at neural measures. Based on a control-systems framework, I predicted that PA and NA biases would be observed over the right and left hemisphere, respectively. The hypothesis was partially supported by the correlation between ventral lateral prefrontal cortex (VLPFC) and individual differences in PA.

VLPFC was implicated across studies as a correlate of positive affect, positive self-appraisal bias and depression severity. VLPFC was also enhanced in trials of the self-evaluation task that involved socially desirable, or “good” words. In C3, a non-linear relationship was identified between VLPFC and symptom severity. An inflection point in neural activity occurred at the threshold for clinical significance in the symptom profile. I briefly explore these results relative to research on intentional emotion regulation. I speculate that VLPFC effects might reflect the active suppression of affective motivation, particularly positive affect. I suggest that PA suppression might be adaptive in the context of C2 functions (i.e., stimulus evaluation), but not in the context of C3 functions (i.e., updating of neural schema). I speculate that symptoms of depression might be related to the over-suppression of positive affect at this stage of the decision-making epoch.

In the last section of this chapter, I consolidate findings across experiments and consider the functional significance of each latent factor. C1 appears to be a stereotyped response to visual stimuli. C2 might reflect the evaluation of word stimuli relative to expectations (i.e., internal schema). In C2, the ventral cortico-limbic system appears dominant. C3 appears related to memory consolidation and the updating on schema based on the evaluative event. In C3, the dorsal cortico-limbic system appears dominant. C4 might reflect sustained attention and emotion regulation. These tentative results suggest that the latent factors are meaningful. The analytic strategies developed herein might be used in future research to test network-oriented hypotheses and models neural self-regulation in the frontal lobe.

Control-Systems Framework for Interpreting Results

In this research, participants consistently showed a positive self-evaluation bias. On average, participants in the child and adolescent sample engaged in positive self-evaluation on 73% of the trials. In two separate samples of undergraduate young adults, positive self-appraisal occurred in 72% and 75% of trials, respectively. Across all studies, measures of trait mood predicted individual differences in evaluative behavior. As predicted, positive bias was decreased in a sample of depressed adults (65%), compared to a non-depressed, “control” group (79%). Results also showed a non-linear relationship between evaluation and symptom severity; the decline in positivity bias was better predicted by changes in the symptom profile when depression was more severe.

This non-linear relationship observed between positive self-evaluation bias and depression severity could be interpreted as a shift in the neural dynamics of frontal lobe self-regulation. A model of frontal lobe functioning posits reciprocal, affect-biased, control systems (Tucker & Luu, 2012; Waters & Tucker, 2013a). The dorsal cortico-limbic system is motivated by positive affective-arousal. It provides a projectional, ballistic mode of operation. In both the motor and psychological domain, this impetus to action is based on internal need states and guided by slow learned (i.e., experience dependent), predictive schema. The ventral cortico-limbic system is motivated by negative affective-arousal. It provides a vigilant mode of operation that focuses attention outward and drives rapid learning with feedback from sensory events. Cognition is a process that emerges from this affective dynamic. As depression becomes more severe,

there may be less dominance of the neural systems that are responsive to external events. Instead, the cognitive process is biased inwardly and reactivity to environmental variance is decreased. This is consistent with observations of increased self-focus in depression (Rimes & Watkins, 2005), as well as disengagement from activities (MacPhillamy & Lewinsohn, 1974) and, in severe cases, a decline in reactivity to emotional stimuli (Tucker, Luu, Frishkoff, et al., 2003).

A control-systems model of self-regulation in the frontal cortex also provides a framework for translating valuable psychological theories into the neural milieu. For example, it suggests several hypothetical routes from transient sadness to lasting and maladaptive depression. Transient depressive symptoms may reflect an adaptive response to loss (e.g., a social exclusion event; DeWall & Baumeister, 2006). Adaptation might involve suppression of positive affective-arousal in the dorsal cortical-limbic path, limiting motivation for projective behavior. When events are consistently aversive, a more stable bias in frontal lobe dynamics could be a result of learning (i.e., learned helplessness, kindling hypothesis; Maier & Seligman, 1976; Monroe & Harkness, 2005). Alternatively, an individual might have a genetic predisposition that makes it difficult to alter this suppressive bias once a shift in the dynamic has occurred (i.e., stress diathesis; Monroe & Simons, 1991). Depressive states are also induced when affective arousal is mechanically or chemically disrupted in the brain, independent of experience dependent biases (i.e., neurological depression; Blumer & Benson, 1975; Mayberg, 1997).

Frontal Lobe Mechanics Instructing Treatment for Depression

The neural mechanics of psychological and psychiatric treatment can also be explored within a control-systems framework. The results of this research suggest an inflection point at which cognitive bias is more closely tied to somatic complaints. Effective behavior therapies address these dysfunctional thinking patterns indirectly by instead promoting engagement in activities, regardless of the patient's motivation to do so (Jacobson, Martell, & Dimidjian, 2001). It may be that this sensory and motor engagement with the environment necessitates a shift in frontal lobe control biases. The lasting impact of continued behavioral activation is that cognitive processes, including the activation of self-schema, must assimilate and accommodate a perfusion of new information (Waters & Tucker, 2013a).

It is notable that the ventral cortical-limbic system of the frontal lobe is preferentially attentive to the environment. The affective-arousal bias in the ventral system is inherently anxious, motivating sensory vigilance and the testing of predictive schema. This is consistent with the frustration-learning hypothesis that combines neural and behavioral evidence to show that negative affect is essential to adaptive learning (Tucker & Luu, 2007; Waters & Tucker, 2012). It follows that an anxious response to grief, loss or surprising non-reward might represent an adaptive shift in the frontal lobe dynamic. Similar to behavior activation therapy, anxiety that is co-morbid with mild or moderate depression may sometimes represent compensatory motivation to attend outward.

Psychopharmacological treatments for depression also alter the control dynamics of the frontal lobe by enhancing the availability of certain neurotransmitters. Although the mechanism of action is complex and largely unknown, the first-line of antidepressants target serotonin and norepinephrine systems that innervate dorsal affective-arousal systems, in particular (Ressler & Nemeroff, 2000). Functions motivated by positive affect are thereby enhanced, and patients are consequently more alert and interested in their surroundings. A similar effect is reported in cases of severe depression following deep brain stimulation (DBS) of frontal limbic regions (Holtzheimer & Mayberg, 2011). A less invasive neural stimulation treatment, transcranial magnetic stimulation (rTMS), instead applies the rhythmic electrical current to the dorsal cortices of the frontal lobe. The current is thought to balance asymmetry in the dorsal system (Teneback et al., 1999). More effective than rTMS is electroconvulsive shock therapy (ECT). ECT sends a current through the frontal lobe, and likely overrides biases in the control system dynamic (Sackeim et al., 1996). As the patient recovers, some of the dysfunctional control biases have been “reset” and the cortex is potentiated for change. Immediately following electrical stimulation with cognitive behavioral therapies could promote lasting change.

Observed at the systems level, these frontal lobe dynamics suggests testable hypotheses related to the onset and maintenance of depression, co-morbid anxiety and depression, as well as the mechanisms of change in treatment. An obstacle to advancing this research area is that empirical support for systems-level theory is largely derived

from studies of isolated neural phenomena (e.g., P300). Efforts to measure network functionality, as in the current research, are a recent priority.

Investigating the Neural Mechanisms of Affective Cognition

Two methodological innovations were central to the current research: the neuropsychometric approach to ERP analysis and the application of principal components analysis (PCA) to the study of network dynamics. Both strategies were adapted from psychometric methodology, which has been productive in describing latent characteristics of affective experience from patterns of covariance. Furthermore, psychometric methods are uniquely applicable to both exploratory research and to the development of applied measurement tools. Though beyond the scope of current knowledge, this could also be a goal of clinical neuroscience: to develop brain-based metrics of individual differences in neural constructs. Exploratory research into the self-regulatory dynamics of the frontal lobe may be further advanced with early orientation toward this goal.

The neuropsychometric approach investigated the neural effects of PA and NA mood dimensions in the word stimuli of a self-evaluation task (Waters & Tucker, 2013b). Clinical research suggests that patterns of reactivity to emotional stimuli might help to differentiate features of mood and anxiety disorders, but findings are mixed (Fitzgerald et al., 2008). A standard approach to testing effects of stimulus valence is to divide stimuli into two groups (good and bad) and then average across all trials in each category. In doing so, variance in the neural signal related to unique affective attributes of each word is lost. Second, it is unlikely that the valence dimension (good-bad) reflects patterns of affective arousal in neural systems. It may be more accurate to align stimulus attributes with PA and NA. These reflect the affective biases in the perceptive mechanism, the brain.

Using a neuropsychometric approach, single trial variance in the psychometric properties of PA and NA was used to generate average ERPs. The central tendency of PA and NA was observed in weighted-average waveforms. Weighted averages showed a differentiation in PA and NA at 400 ms in anterior channels, coincident with the MFN waveform. These results suggest that the cognitive process involves evaluation of stimulus affectivity at that time. Overall, effects of psychometric weighting showed NA larger than PA, as if the calm-anxious dimension better characterized the affective nature

of the self-evaluation task. Finally, the neuropsychometric approach revealed EEG features that separated dorsal-posterior cortex and ventral-anterior cortex. Consistent with the control-systems framework, this whole-brain effect might reflect reciprocal network dynamics in the decision-making epoch (Waters & Tucker, 2013b).

Factor Analysis in ERP Research

To further investigate this phenomenon, I again borrowed from psychometric methodology. In the study of mood dimensionality, factor analysis was used to define constituent parts of emotional experience, PA and NA (Watson & Tellegen, 1985). In ERP research, factor analysis is instead used to clean EEG data, or to study an ERP waveform in isolation (Dien & Frishkoff, 2005). Following the methods of psychometric research, I instead used factor analysis to investigate the latent factor structure of the decision-making ERP. I hypothesized that the time course and whole-brain topography of latent factors would be consistent with the reciprocal action of limbic-cortical control systems. Relative to an affective-arousal model of neural self-regulation, I further hypothesized that factors might reflect shifting dominance between PA and NA bias, and thus differentiate depressed from neurotypical responses.

As this was an exploratory investigation, my hypotheses and strategy were guided by a previous application of principal components analysis (PCA) to a series of ERPs that are relevant to mood-cognition interaction in decision-making. Among this series is the classic P300, followed the late positive potential (LPP). The P300 and LPP ERPs are modulated by stimulus valence and trait affect (J. Polich, 2007). The LPP is also modulated by intentional emotion regulation (G. Hajcak & Nieuwenhuis, 2006). An early constituent of the posterior positivity, the P1r, has received less attention, but also appears to be modulated by affect (Waters & Tucker, 2013b). A challenge in this research is that the P1r, P300 and LPP are superimposed in time and topography, summing to produce a wide, positive going ERP over the parietal cortex. Very recently, a handful of investigators have used PCA to decompose the posterior positivity into constituent parts (Greg Hajcak, Dunning, & Foti, 2009). The focus of this research, however, remains isolated on one or another ERP.

The current research builds on this recent effort by widening the context of the PCA decomposition to the study of whole brain dynamics. As predicted, the P1r, P300

and LPP were divided into the largest components of a seven-factor structure (i.e., accounting for the most variance). This pattern replicated across three separate studies, as did the general topography of each component (Figures 20-21). The latent factor structure described correlated activity on the entire scalp surface; grouping the classical average ERPs that are commonly studied in isolation. Importantly, this topography included both posterior and anterior effects, which are of particular importance to depression research.

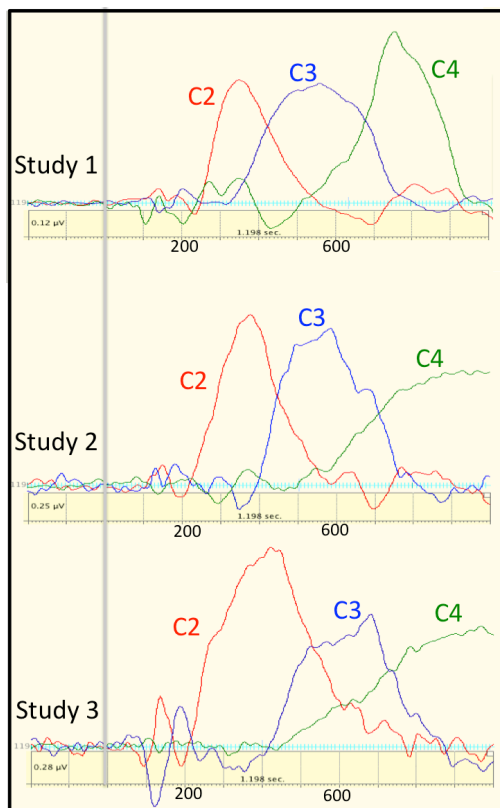


Figure 20. Replication of factor structure across three studies. Time scale in ms, stimulus onset at grey bar. All waveforms at Pz (or channel 119).

Depression Neuropathology in Self-Evaluative Cognition

The frontal lobe functions are essential in decision-making, social cognition, and affective self-regulation. This circuitry may be altered in depression, as evidenced by predictable changes in self-evaluative behavior. A secondary goal of this research was to characterize effects of depression in the latent factor structure. Findings place isolated

ERP components in a functional context and provide a preliminary measure of whole brain dynamics that may be pathological in clinical populations. The components analysis characterized effects of depression with striking specificity. C2 was exaggerated in individuals reporting more severe and frequent symptoms of depression. C3, in contrast, was attenuated in this group. The time course and topography of these components may differentiate anxious and depressed affective biases in the clinical sample.

C2 captures the variance of the MFN, a well-studied average ERP, and a posterior correlate, the P1r. The MFN is part of a suite of medial frontal negativities that are responsive to discrepant events, negative feedback and errors (Phan Luu et al., 2000). It is exaggerated in clinical anxiety and high NA. The P1r retraces some of the visual pathways following frontal lobe processing of stimuli. Given what is known about these two ERP components, C2 appears involved in stimulus processing. In anxious-depression, it may also capture an exaggerated negative response to word stimuli.

In C3, variance in the P300 is correlated with an anterior positivity. The P300 is associated with memory functions and updating of the context (i.e., schema) following an event (Donchin & Coles, 1988). P300 amplitude is decreased in depression and low PA (Polich, 1998). It is larger in yes-good trials and correlates with the positivity bias in memory recall (L. A. Watson et al., 2007). C3 appears to index functions related to the neural assimilation of information that has been drawn from the evaluative event. It may be that in depression, there is less adaptation of internal schema (including self-schema), particularly in response to rewarding (i.e., positive) events.

Affective Dimensionality and Self-Evaluative Behavior

Further consideration of comorbid anxiety and depression was addressed in behavioral measures of self-evaluation. I tested a dimensional specificity hypothesis in self-evaluative behavior: that trait PA scores will predict responses to desirable words, and trait NA scores will predict responses to undesirable words. In the child and adolescent sample as well as the clinical sample, this hypothesis was supported; PA was uniquely predictive of responses to positive words, and NA was uniquely predictive of responses to negative words. The decline of positive endorsement in depression, however, was equally attributable to behavior change on good- and bad-word trials. In Study 2, support for the hypothesis was modest; both PA and NA were predictive of

either word type but effect sizes were consistent with the hypothesized pattern. In Study 3, only PA predicted endorsement behavior involving either word type. Taken together, results suggest that the PA scale is most consistently related to self-appraisal and that the hypothesized pattern might only appear in clinical samples and youth.

This line of inquiry is significant because it suggests that the decline in positivity bias involves more rejection of desirable traits in depression, and more endorsement of negative traits in anxiety. To the extent that this distinguishes clinical presentations and generalizes beyond the psychometric task, it may also instruct the development of targeted therapeutic activities. In future research, convergent measures of trait affect should be used to reduce the redundancy in the bivariate measure. Using the current method, results might simply reflect a replication of Tellegan and Watson's (1985) initial observation of PA and NA factors in emotional experience. It follows that so classifying word stimuli on the self-evaluation task would enhance the predictive nature of a PA and NA summary score. It may be most productive, however, to test the hypothesis in a naturalistic setting where "rejection of the desirable" and "acceptance of the undesirable" can be operationalized in a broader behavioral repertoire.

Neural Correlates of Self-Regulation in RVL PFC

In addition to characterizing affect-cognition interaction in behavioral measures, I identified neural correlates of mood and self-evaluation within the latent factor structure. Right ventral lateral prefrontal cortex (RVL PFC) was consistently implicated. In C3, the component attenuated in the depression, the relationship between symptom severity and RVL PFC intensity was non-linear; intensity increased with mild to moderate symptoms but then decreased in individuals who reported severe and frequent symptoms. As in the behavioral data, there appeared to be an inflection point in the sample that implied a categorical shift in frontal lobe dynamics.

It may be that, as symptoms become clinically significant, there is a transition in the neural self-regulation dynamic of the frontal cortex. One interpretation is that in neurotypical functioning, the regulatory action of RVL PFC increases with a decline in positive mood. But in clinically significant depression, RVL PFC is increasingly withdrawn from emotional events. A similar shift has been observed in medial frontal response to negative feedback; the MFN is exaggerated in mild depression and anxiety,

but then declines with more severe and frequent symptoms (Tucker, Luu, Frishkoff, et al., 2003).

Results of Study 2, however, were not consistent with this interpretation. Instead, the neural response of non-depressed, young adult participants replicated those observed in the more severely depressed sample; RVL PFC negativity on the scalp surface decreased as trait PA decreased. Study 2 also showed that the effect was not specific to self-evaluation; effect sizes in the other-evaluation task may even be larger. However, additional evidence for RVL PFC engagement with positive emotional experience appeared in the valence contrast. Good word trials evoked a larger negativity in RVL PFC than did bad word trials. This effect was particularly robust in C2, where it also replicated in Study 3. In this separate sample of non-depressed young adults, however, the linear association between RVL PFC and mood was absent.

Though inconsistent across studies, the RVL PFC effect is notable. First, it addressed the hypothesis posited by the affective-arousal model that left and right hemispheres show an NA and PA bias, respectively. PA (not NA) correlates were identified over right hemisphere (not left hemisphere). Second, it is consistent with evidence from other research domains that implicates RVL PFC in emotion regulation, and the regulation of positive affect, in particular. For example, the most consistent injury producing secondary mania (pathologically high PA) is a lesion of the right ventrolateral frontal or basal temporal cortex (Starkstein, Fedoroff, Berthier, & Robinson, 1991). RVL PFC metabolism has been shown to increase when individuals engage cognitive strategies to suppress an emotional response (Lieberman et al., 2007). Some have shown that this suppression effect is specific to positive emotional experiences (Light et al., 2011). Enhanced RVL PFC activity during positive affect suppression was also predictive of poor treatment outcome in depressed individuals, as if the pathology was related to over-suppression of positive affect. A recent analysis of tractography data by Mayberg and colleagues showed that integrity of RVL PFC connectivity (greater local and reduced global connectivity) is associated with resiliency in individuals at risk for depression (Cisler et al., 2013).

If RVL PFC activity represents the suppression of positive affect, interpretation of the current finding would be paradoxical. Results show more RVL PFC activity (i.e.,

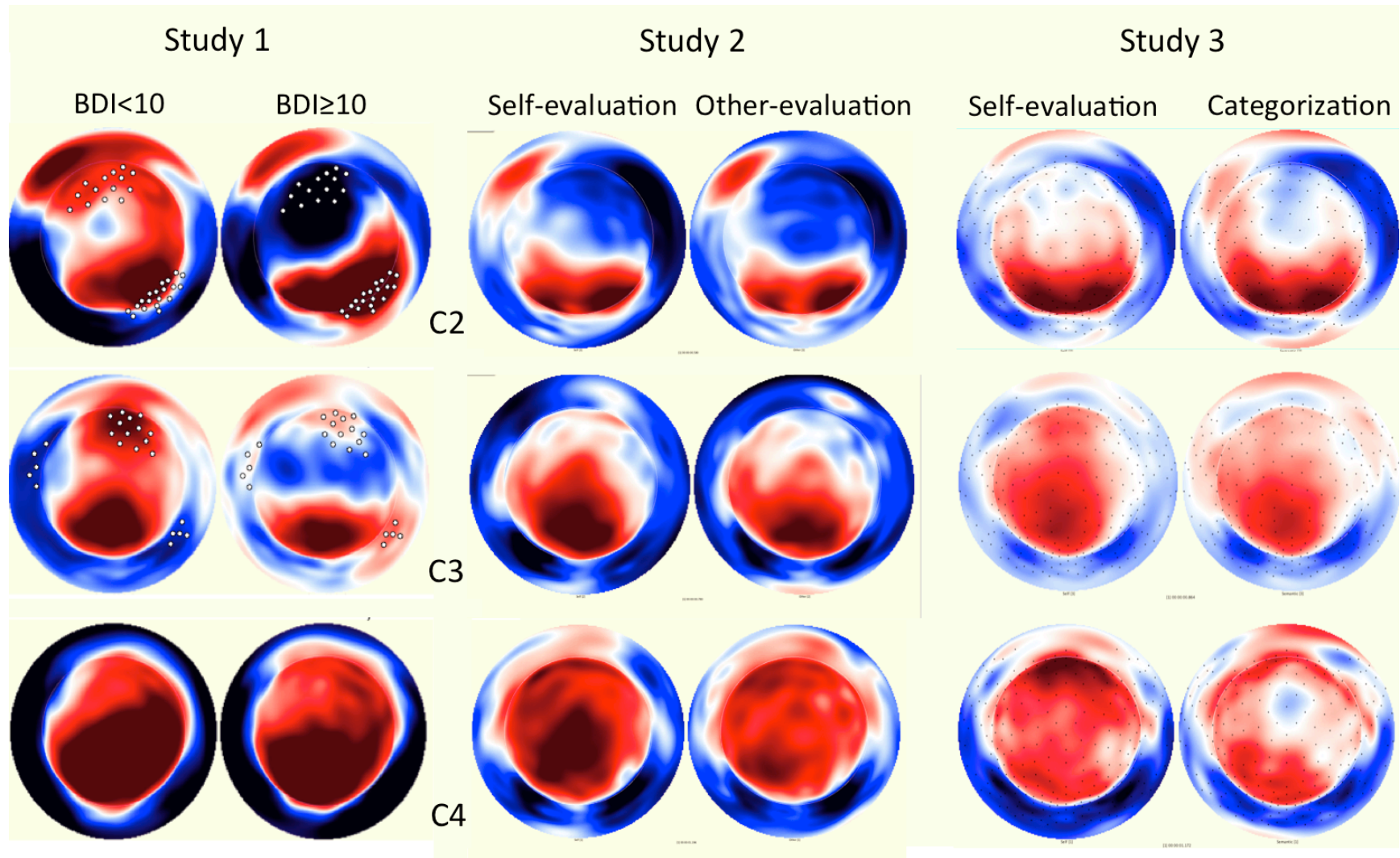
more suppression) with increasingly positive experiences (e.g., positive words, higher PA, lower BDI scores). However, time course and topography of the latent factor structure offers some compelling context for speculation (Figure 21). C2 is involved in appraising the extent to which external events deviate from expectations (i.e., schema), and shows an NA bias. It may be that RVL PFC suppression of PA is adaptive in C2 functioning, and therefore more robust in persons who present a strong PA bias. C3, in contrast, reflects the updating internal schema following an informative event. It may be that cortical control of PA should be released at this stage of the decision-making epoch, as encoding processes require engagement of the dorsal, slow-learning system. Importantly, RVL PFC suppression continued in C3 for depressed study participants. This may reflect dysfunction in the reciprocal dynamic of control systems, such that impairment in C3 functions contribute to a stable decrease in positive self-evaluation bias.

Tentative Interpretation of the Component Structure

Component 1: stereotyped visual response. Across studies, C1 captured variance in the stereotyped visual response (i.e., visual P1, visual N1, frontal P2) and was the fourth factor (i.e., explained less variance than C1-C3). This early component preceded the posterior positivity in the decision-making ERP and was therefore not a focus of analyses.

Component 2: evaluation of word stimuli. C2 reflects dominance of ventral cortical limbic characteristics including anxious affective bias and cognitive processing of stimuli. Effects of emotion variables were paradoxical, suggesting some reciprocity between dorsal and ventral networks in the suppression of positive affect.

Figure 21. (next page) Topography of component amplitude across studies.



Across studies, C2 reaches a maximum in a time window previously associated with effects of word valence and endorsement decisions. In posterior channels, C2 captured the P1r, an early component of the posterior positivity. The P1r recapitulates a visual response as if engaged in re-entrant stimulus processing. Correlated activity in anterior channels of C2 captured variance in the MFN, an average ERP waveform associated with the detection of discrepancy from expectation, error self-monitoring and feedback monitoring. An exaggerated MFN has been associated with anxiety (high NA) and moderate depression, but appears attenuated in severe depression (Phan Luu et al., 2000; Tucker, Luu, Frishkoff, et al., 2003). Taken together, these characteristics strongly associate C2 with the evaluative portion of the decision-making ERP.

C2 topography appears asymmetric in the ventral lateral frontal lobe: positive going on the left and negative going on the right. The negativity over right ventral lateral prefrontal cortex (RVLPFC) was consistently associated with positive emotional variables: enhanced in response to good words and self-evaluation, positively correlated with PA in the healthy control samples, and negatively correlated with BDI in the clinical sample. Correlates of positive self-evaluation bias also followed this pattern, implicating bilateral anterior ventral cortices, as well as the right rostral anterior cingulate cortex.

Paradoxically, metabolic and neurological evidence suggests the hypothesis that RVLPFC activity is greater during positive affect suppression. Yet these results show increased activity with positive emotional behavior and variables. The context of word stimulus processing suggests an interpretation; C2 might capture ventral constraint on dorsal biases. Indeed, stimulus processing is an inherently anxious event that would require suppression of the projective mode, motivated by positive affect. In adaptive functioning (e.g., evaluation of a visual stimulus) this active suppression effect might be stronger in persons with higher trait positive affect.

Contrary to this hypothesis, however, C2 amplitude was exaggerated in depression over anterior medial, and right posterior cortex. This might be an effect of co-morbid anxiety present in the clinical sample. The effect was strong, as if participants with clinically significant symptoms were more anxiously engaged in stimulus processing. But this exaggerated effect in the clinical sample is inconsistent with results of the correlation analysis, which associated exaggerated C2 features with increasing PA

and positive valence. To understand this paradoxical effect, it may be helpful to recognize that group mean differences in scalp amplitude may be independent of linear patterns (i.e., in VLPFC). The implication of these scalp findings, it follows, is that the linear effects in RVL PFC of depressed-anxious individuals might take place in a fundamentally altered milieu; one that begins with an exaggerated suppression of dorsal functions. Similarly, amplified indices of stimulus processing in the clinical sample might represent a compensatory drive to override the apathy of low positive affect. In depression, it may take more of C2 to engage in the task.

Component 3: updating self-schema in the central midline. C3 reflects a shift in stimulus processing from evaluation to neural encoding of the event. This memory-related process is better associated with dorsal functions; slow learning networks of the dorsal midline (Waters & Tucker, 2012). Across studies, C3 was maximal within the time window of the classic P300 of the average ERP. The P300 is related to attention and memory encoding (Donchin & Coles, 1988). It is sensitive to events that provide information relative to a motivational context. From a control-systems perspective, learning and memory represent the ongoing process of developing and adjusting predictive models, or neural schema. Although the self-evaluation task in this experiment is removed from typical social behavior, the process still involves projection and updating of a self-schema. Though effects were subtle, self-evaluation was greater than other evaluation along the midline in Study 2, particularly over the central midline. In Study 3, condition effects were again oriented along the midline in C3; self-evaluation was greater than the categorization task, particularly over anterior midline. Research on the neural basis of self, and self-reflective cognition consistently implicates the midline cortical structures (Qin & Northoff, 2011).

In Study 2, more robust effects of valence (good>bad) were also oriented along the midline. This valence effect has been previously reported in the context of self-evaluation. Since P300 is sensitive to motivational context, this effect is thought to reflect the positivity bias in self-evaluation. Relevant to this, C3 was attenuated in Study 1 for participants with moderate to severe symptoms of depression. Furthermore, C3 was the factor that accounted for the most variance in Study 1. In Study 2 and Study 3, however,

C3 was the smallest of the first four components. Taken together, these observations suggest an relationship of C3 with depression neuropathology.

The correlation analysis, however, complicates the picture. In Study 2, C3 showed some evidence of an effect that was robust in C2: higher PA and positive words were associated with greater activity in RVL PFC. For depressed participants in Study 1, RVL PFC was also greater with more engagement in positive self-evaluation. Unlike C2, however, C3 showed a non-linear of BDI scores and RVL PFC activity; for depressed participants, the inverse correlation of RVL PFC and symptom severity remained consistent with C2. For non-depressed participants, however, the pattern reversed such that RVL PFC activity was the least in those reporting the fewest symptoms. I previously speculated that RVL PFC activity was related to positive affect suppression. This finding suggests that in C3, it may be adaptive to release inhibition on positive affect in order to facilitate encoding processes in the dorsal cortex. Consistent with this conceptualization, the low symptom group showed no correlation between self-evaluation bias and VLPFC. Instead, self-evaluation bias was associated with visual cortex. These effects may reflect an adaptive part of the consolidation process; a shift from ventral-anterior stimulus processing to dorsal-posterior encoding of the event. It may be that in depression, C3 functions are disrupted by pathology in the shift from PA suppression to dominance of the dorsal network.

Component 4: sustained attention. Across studies, C4 captured variance in the LPP, of the average ERP. The LPP responds to emotional and arousing stimuli (G. Hajcak et al., 2010). It is also reduced by voluntary regulation of negative emotion (G. Hajcak & Nieuwenhuis, 2006). There is some evidence that the LPP is only present with conscious awareness of a stimulus. Taken together, C4 may represent a state of sustained attention to the affective quality of an event that serves as a volitional or innate form of emotion regulation.

Consistent with this hypothesis, self-evaluation was greater than semantic-categorization over dorsal anterior cortex, as if additional attention resources were recruited following social appraisal. Valence effects showed a similar anterior distribution (good>bad) as well as a left posterior effect (bad>good). Particularly over the

frontal cortex, these effects might relate to features of the study paradigm designed to extend and deepen the evaluative experience of participants.

In Study 2 and Study 3, I decreased the timed nature of the task by cueing responses 1 second after the onset of the word stimulus. I also altered the responses from a dichotomous option (*like me, not like me*) to a four-point scale. These adaptations of the experimental design were intended to increase the introspective nature of the task, and decrease the simple valence judgment. They may have also had a telling impact on the C4 structure and topography. Most notably, a focal anterior positivity shifts to C4 in Study 2 and Study 3. The wave shape of C4 also changed such that it extends beyond the analytic window. Finally, of the first four factors in Study 1, C4 explained the least amount of variance. In Study 2 and 3, however, C4 was the largest factor, capturing most of the variance in the sample. It may be that this variance was generated by a somewhat introspective delay before the response. It is as if some attention-related resources have shifted forward in the time course of the decision-making ERP. Future research might explore the hypothesis that C4 will be exaggerated as a function of ruminative behavior.

Conclusions

Across a series of three studies, measures of mood state predicted positive self-evaluation bias. Positive bias was decreased in depressed individuals, and there appears to be an enhancement of the mood-cognition relationship in the context of clinically significant symptoms. Activity in right ventral lateral prefrontal cortex was identified as a neural correlate of trait positive affect. This predictive relationship might reflect positive affect suppression. This may be adaptive through evaluative stages of the decision-making epoch, but maladaptive in subsequent encoding stages.

As hypothesized, statistical decomposition separated a posterior positivity in three components. The pointed and round features previously identified with the neuropsychometric approach were also observed in separate component waveforms. A goal of this research was to align latent factors of the evaluative ERP with a model of neural self-regulation. Germane to this goal, the factor structure was replicated in time course and topography across three independent studies.

C2 appeared integral to the evaluation of word stimuli; a stage at which semantic meaning is compared with affect-biased expectations. Asymmetric effects in ventral

lateral prefrontal cortex suggested preferential engagement of the ventral cortico-limbic system. It follows that exaggeration of C2 in the depressed sample might reflect co-morbid anxiety, which is adaptive insofar as it promotes engagement in the evaluative task. However, instead of an NA bias, correlates of PA were detected in C2. Interpreted as PA suppression, the effect might also represent adaptive emotion regulation in service of task engagement. C3 may be involved in the updating of neural schema following the evaluative event. The topography of this component was most consistent with dorsal cortico-limbic system. Evidence supports the hypothesis that PA suppression is not adaptive in C3, and may represent a locus of pathology in depression. C4 appears to reflect sustained attention following the evaluative, and intrinsic management of affect-biased dynamics following an emotional effect.

Though speculative, this synthesis suggests testable hypotheses for future research. An advantage of observing ERP phenomena within a factor structure is that it allows for a whole brain decomposition of temporally distinct processing stages. This exploratory analysis may be the first to observe the decision-making epoch in putative measures of EEG network dynamics. Findings demonstrate the potential in convolving psychometric methods with brain imaging research to measure individual differences in neural dynamics.

REFERENCES CITED

- American Psychiatric Association. (1994). *Diagnostic and Statistical Manual (DSM-IV)*. Washington, D.C.: American Psychiatric Association.
- Barbas, H., & Pandya, D. (1989). Architecture and intrinsic connections of the prefrontal cortex in the rhesus monkey. *Journal of Comparative Neurology*, 286(3), 353-375.
- Barrett, L. F., & Wager, T. D. (2006). The structure of emotion evidence from neuroimaging studies. *Current Directions in Psychological Science*, 15(2), 79-83.
- Beck, A., Steer, R., & Brown, G. (1996). Manual for the BDI-II: San Antonio, TX: Psychological Corporation.
- Beck, A. T., & Steer, R. A. (1990). *Beck Anxiety Inventory: BAI; manual*: Psychological Corporation, Harcourt Brace Jovanovich.
- Berridge, K. C., & Scherer, K. (2003). Comparing the emotional brains of humans and other animals. *Handbook of affective sciences*, 25-51.
- Bloom, J. S., & Hynd, G. W. (2005). The role of the corpus callosum in interhemispheric transfer of information: excitation or inhibition? *Neuropsychology Review*, 15(2), 59-71.
- Blumer, D., & Benson, D. F. (1975). Personality changes with frontal and temporal lobe lesions. *Psychiatric Aspects of Neurologic Disease*, 1, 151-170.
- Bradley, M. M., Sabatinelli, D., Lang, P. J., Fitzsimmons, J. R., King, W., & Desai, P. (2003). Activation of the visual cortex in motivated attention. *Behavioral Neuroscience*, 117(2), 369.
- Briesemeister, B. B., Tamm, S., Heine, A., & Jacobs, A. M. (2013). Approach the Good, Withdraw from the Bad; Review on Frontal Alpha Asymmetry Measures in Applied Psychological Research. *Psychology*, 4, 261.
- Brown, J. D., & Mankowski, T. A. (1993). Self-esteem, mood, and self-evaluation: changes in mood and the way you see you. *Journal of Personality and Social Psychology*, 64(3), 421.
- Brown, T. A., Chorpita, B. F., & Barlow, D. H. (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*, 107(2), 179.

- Bruder, G. E., Tenke, C. E., Warner, V., Nomura, Y., Grillon, C., Hille, J., . . . Weissman, M. M. (2005). Electroencephalographic measures of regional hemispheric activity in offspring at risk for depressive disorders. *Biological Psychiatry*, 57(4), 328-335.
- Buzsaki, G., & Watson, B. O. (2012). Brain rhythms and neural syntax: implications for efficient coding of cognitive content and neuropsychiatric disease. *Dialogues in Clinical Neuroscience*, 14(4), 345.
- Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion*, 4(1), 3.
- Cisler, J., James, G., Tripathi, S., Mletzko, T., Heim, C., Hu, X., . . . Kilts, C. (2013). Differential functional connectivity within an emotion regulation neural network among individuals resilient and susceptible to the depressogenic effects of early life stress. *Psychological Medicine*, 43(3), 507-518.
- Clark, L. A., & Watson, D. (1991). Tripartite model of anxiety and depression: psychometric evidence and taxonomic implications. *Journal of Abnormal Psychology*, 100(3), 316.
- Coan, J. A., & Allen, J. J. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, 67(1), 7-50.
- Coan, J. A., Allen, J. J., & McKnight, P. E. (2006). A capability model of individual differences in frontal EEG asymmetry. *Biological Psychology*, 72(2), 198-207.
- Codispoti, M., Ferrari, V., & Bradley, M. M. (2007). Repetition and event-related potentials: distinguishing early and late processes in affective picture perception. *Journal of Cognitive Neuroscience*, 19(4), 577-586.
- Courchesne, E., Hillyard, S. A., & Galambos, R. (1975). Stimulus novelty, task relevance and the visual evoked potential in man. *Electroencephalography and Clinical Neurophysiology*, 39(2), 131-143.
- Craik, F. I., Moroz, T. M., Moscovitch, M., Stuss, D. T., Winocur, G., Tulving, E., & Kapur, S. (1999). In search of the self: A positron emission tomography study. *Psychological Science*, 10(1), 26-34.
- Crawford, J. R., & Henry, J. D. (2004). The Positive and Negative Affect Schedule (PANAS): Construct validity, measurement properties and normative data in a large non-clinical sample. *British Journal of Clinical Psychology*, 43(3), 245-265.
- D'Argembeau, A., Collette, F., Van der Linden, M., Laureys, S., Del Fiore, G., Degueldre, C., . . . Salmon, E. (2005). Self-referential reflective activity and its relationship with rest: a PET study. *Neuroimage*, 25(2), 616-624.

- Damasio, A. (2012). *Self comes to mind: Constructing the conscious brain*. NY, NY: Random House.
- Damasio, H., Grabowski, T., Frank, R., Galaburda, A. M., & Damasio, A. R. (1994). The return of Phineas Gage: clues about the brain from the skull of a famous patient. *Science*, 264(5162), 1102-1105.
- Davidson, R. J. (1992). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition*, 20(1), 125-151.
- Davidson, R. J. (1998). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition and Emotion*, 12(3), 307-330.
- Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Sciences*, 3, 11-21.
- DeWall, C. N., & Baumeister, R. F. (2006). Alone but feeling no pain: Effects of social exclusion on physical pain tolerance and pain threshold, affective forecasting, and interpersonal empathy. *Journal of Personality and Social Psychology*, 91(1), 1.
- Dien, J. (2010). The ERP PCA Toolkit: An open source program for advanced statistical analysis of event-related potential data. *Journal of Neuroscience Methods*, 187(1), 138-145.
- Dien, J., & Frishkoff, G. A. (2005). Introduction to Principal Components Analysis of Event-Related Potentials. In H. T (Ed.), *Event Related Potentials: A Methods Handbook*. Cambridge: MIT Press.
- Donchin, E., & Coles, M. G. (1988). Is the P300 component a manifestation of context updating? *Behavioral and Brain Sciences*, 11(03), 357-374.
- Drevets, W. C., Price, J. L., & Furey, M. L. (2008). Brain structural and functional abnormalities in mood disorders: implications for neurocircuitry models of depression. *Brain Structure and Function*, 213, 93-118. doi: 10.007/s00429-008-0189-x
- Dunbar, R. I., & Shultz, S. (2007). Evolution in the social brain. *Science*, 317(5843), 1344-1347.
- Duncan-Johnson, C. C., & Donchin, E. (1977). On quantifying surprise: The variation of event-related potentials with subjective probability. *Psychophysiology*, 14(5), 456-467.
- Ekman, P., & Friesen, W. V. (1971). Constants across cultures in the face and emotion. *Journal of Personality and Social Psychology*, 17(2), 124-129.

- Esslen, M., Metzler, S., Pascual-Marqui, R., & Jancke, L. (2008). Pre-reflective and reflective self-reference: a spatiotemporal EEG analysis. *Neuroimage*, 42(1), 437-449.
- Etkin, A., & Wager, T. (2007). Functional neuroimaging of anxiety: a meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *American Journal of Psychiatry*, 164(10), 1476-1488.
- Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological Methods*, 4(3), 272.
- Fields, E. C., & Kuperberg, G. R. (2012). It's all about you: An ERP study of emotion and self-relevance in discourse. *Neuroimage*, 62(1), 562-574.
- Fitzgerald, P. B., Laird, A. R., Maller, J., & Daskalakis, Z. J. (2008). A meta-analytic study of changes in brain activation in depression. *Human Brain Mapping*, 29, 683-695. doi: 10.1002/hbm.20426
- Gable, P., & Harmon-Jones, E. (2010). The motivational dimensional model of affect: Implications for breadth of attention, memory, and cognitive categorisation. *Cognition and Emotion*, 24(2), 322-337.
- Gainotti, G. (1989). Disorders of emotions and affect in patients with unilateral brain damage. *Handbook of Neuropsychology*, 3, 345-361.
- Gencoz, & Tulin. (2002). Discriminant validity of low positive affect: is it specific to depression? *Personality and Individual Differences*, 32(6), 991-999.
- Glenn Northcutt, R., & Kaas, J. H. (1995). The emergence and evolution of mammalian neocortex. *Trends in Neurosciences*, 18(9), 373-379.
- Goldman-Rakic, P. S., Bates, J. F., & Chafee, M. V. (1992). The prefrontal cortex and internally generated motor acts. *Current Opinion in Neurobiology*, 2(6), 830-835.
- Gruber, J., Johnson, S. L., Oveis, C., & Keltner, D. (2008). Risk for mania and positive emotional responding: too much of a good thing? *Emotion*, 8(1), 23.
- Hajcak, G., Dunning, J. P., & Foti, D. (2009). Motivated and controlled attention to emotion: time-course of the late positive potential. *Clinical Neurophysiology*, 120(3), 505-510.
- Hajcak, G., MacNamara, A., & Olvet, D. M. (2010). Event-related potentials, emotion, and emotion regulation: an integrative review. *Developmental Neuropsychology*, 35(2), 129-155.

- Hajcak, G., & Nieuwenhuis, S. (2006). Reappraisal modulates the electrocortical response to unpleasant pictures. *Cognitive Affective Behavioral Neuroscience*, 6(4), 291-297.
- Heatherton, T. F., Wyland, C. L., Macrae, C. N., Demos, K. E., Denny, B. T., & Kelley, W. M. (2006). Medial prefrontal activity differentiates self from close others. *Social Cognitive and Affective Neuroscience*, 1(1), 18-25.
- Herbert, C., Herbert, B. M., Ethofer, T., & Pauli, P. (2011). His or mine? The time course of self-Äother discrimination in emotion processing. *Social Neuroscience*, 6(3), 277-288.
- Herbert, C., Pauli, P., & Herbert, B. M. (2011). Self-reference modulates the processing of emotional stimuli in the absence of explicit self-referential appraisal instructions. *Social Cognitive and Affective Neuroscience*, 6(5), 653-661.
- Holtzheimer, P. E., & Mayberg, H. S. (2011). Deep brain stimulation for psychiatric disorders. *Annual Review of Neuroscience*, 34, 289-307.
- Izard, C. E. (2007). Basic emotions, natural kinds, emotion schemas, and a new paradigm. *Perspectives on Psychological Science*, 2(3), 260-280.
- Jacobson, N. S., Martell, C. R., & Dimidjian, S. (2001). Behavioral activation treatment for depression: Returning to contextual roots. *Clinical Psychology: Science and Practice*, 8(3), 255-270.
- Johnson, S. C., Baxter, L. C., Wilder, L. S., Pipe, J. G., Heiserman, J. E., & Prigatano, G. P. (2002). Neural correlates of self-Äreflection. *Brain*, 125(8), 1808-1814.
- Kelley, W. M., Macrae, C. N., Wyland, C. L., Caglar, S., Inati, S., & Heatherton, T. F. (2002). Finding the self? An event-related fMRI study. *Journal of Cognitive Neuroscience*, 14(5), 785-794.
- Kennedy, D. P., & Courchesne, E. (2008). Functional abnormalities of the default network during self-and other-reflection in autism. *Social Cognitive and Affective Neuroscience*, 3(2), 177-190.
- Kessler, R. C., Akiskal, H. S., & al., e. (2006). Prevalence and effects of mood disorders on work performance in a nationally representative sample of US workers. *American Journal of Psychiatry*, 163, 1561-1568.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 593-602.

- Kessler, R. C., Burglund, P., & al, e. (2003). The epidemiolgy of major depressive disorder: results from teh national comorbidity survey replication (NCS-R). *Journal of American Medical Association*, 289, 3095-3105.
- Kessler, R. C., Chiu, W. T., Demier, O., & Walters, E. E. (2005). Prevalence, severity and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Journal of the American Medical Association*, 62(6), 617-627. doi: 10.1001/archpsyc.62.6.617
- Kiehl, K. A. (2006). A cognitive neuroscience perspective on psychopathy: evidence for paralimbic system dysfunction. *Psychiatry Research*, 142(2), 107-128.
- Knyazev, G. G., Savostyanov, A. N., Volf, N. V., Liou, M., & Bocharov, A. V. (2012). EEG correlates of spontaneous self-referential thoughts: a cross-cultural study. *International Journal of Psychophysiology*, 86(2), 173-181.
- Lamers, F., van Oppen, P., Comijs, H. C., Smit, J. H., Spinhoven, P., Van Balkom, A., . . . Penninx, B. (2011). Comorbidity patterns of anxiety and depressive disorders in a large cohort study: the Netherlands Study of Depression and Anxiety (NESDA). *The Journal of Clinical Psychiatry*, 72(3), 341-348.
- Lang, S. F., Nelson, C. A., & Collins, P. F. (1990). Event-related potentials to emotional and neutral stimuli. *Journal of Clinical and Experimental Neuropsychology*, 12(6), 946-958.
- Leary, M. R. (2007). Motivational and emotional aspects of the self. *Annual Review of Psychology*, 58, 317-344.
- Lemongne, C., Mayberg, H. S., Bergouignan, L., Volle, E., Delaveau, P., Lehericy, S., . . . Fossati, P. (2010). Self-referential processing and the prefrontal cortex over the course of depression: A pilot study. *Journal of Affective Disorders*, 124, 196-201. doi: 10.1016/j.jad.2009.11.003
- Lewinsohn, P. M., Solomon, A., Seeley, J. R., & Zeiss, A. (2000). Clinical implications of "subthreshold" depressive symptoms. *Journal of Abnormal Psychology*, 109(2), 345-351.
- Lieberman, M. D., Eisenberger, N. I., Crockett, M. J., Tom, S. M., Pfeifer, J. H., & Way, B. M. (2007). Putting feelings into words affect labeling disrupts amygdala activity in response to affective stimuli. *Psychological Science*, 18(5), 421-428.
- Light, S. N., Heller, A. S., Johnstone, T., Kolden, G. G., Peterson, M. J., Kalin, N. H., & Davidson, R. J. (2011). Reduced right ventrolateral prefrontal cortex activity while inhibiting positive affect is associated with improvement in hedonic capacity after 8 weeks of antidepressant treatment in major depressive disorder. *Biological Psychiatry*, 70(10), 962-968.

- Liu, Y., Sheng, F., Woodcock, K. A., & Han, S. (2013). Oxytocin effects on neural correlates of self-referential processing. *Biological Psychology*, 94(2), 380-387.
- Lovejoy, M. C., & Steuerwald, B. L. (1995). Subsyndromal unipolar and bipolar disorders: Comparisons on positive and negative affect. *Journal of Abnormal Psychology*, 104(2), 381.
- Luu, P., Collins, P., & Tucker, D. M. (2000). Mood, personality, and self-monitoring: negative affect and emotionality in relation to frontal lobe mechanisms of error monitoring. *Journal of Experimental Psychology*, 129(1), 43.
- Luu, P., Flaisch, T., & Tucker, D. M. (2000). Medial frontal cortex in action monitoring. *Journal of Neuroscience*, 20(1), 464-469.
- Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: does happiness lead to success? *Psychological Bulletin*, 131(6), 803.
- MacNamara, A., Foti, D., & Hajcak, G. (2009). Tell me about it: neural activity elicited by emotional pictures and preceding descriptions. *Emotion*, 9(4), 531.
- MacPhillamy, D. J., & Lewinsohn, P. M. (1974). Depression as a function of levels of desired and obtained pleasure. *Journal of Abnormal Psychology*, 83(6), 651.
- Magno, E., & Allan, K. (2007). Self-Reference During Explicit Memory Retrieval An Event-Related Potential Analysis. *Psychological Science*, 18(8), 672-677.
- Maier, S. F., & Seligman, M. E. (1976). Learned helplessness: Theory and evidence. *Journal of Experimental Psychology: General*, 105(1), 3.
- Mayberg, H. S. (1997). Limbic-cortical dysregulation: A proposed model of depression. *Journal of Neuropsychiatry*, 9(3), 471-481.
- Mayberg, H. S. (2003). Modulating dysfunctional limbic-cortical circuits in depression: towards development of brain-based algorithms for diagnosis and optimised treatment. *British Medical Bulletin*, 65, 193-207.
- Miller, G. A. (2010). Mistreating psychology in the decades of the brain. *Perspectives on Psychological Science*, 5(6), 716-743.
- Monroe, S. M., & Harkness, K. L. (2005). Life stress, the "kindling" hypothesis, and the recurrence of depression: considerations from a life stress perspective. *Psychological Review*, 112(2), 417.
- Monroe, S. M., & Simons, A. D. (1991). Diathesis-stress theories in the context of life stress research: implications for the depressive disorders. *Psychological Bulletin*, 110(3), 406.

- Mor, N., & Winquist, J. (2002). Self-focused attention and negative affect: a meta-analysis. *Psychological Bulletin*, 128(4), 638.
- Moser, J. S., Hajcak, G., Bukay, E., & Simons, R. F. (2006). Intentional modulation of emotional responding to unpleasant pictures: an ERP study. *Psychophysiology*, 43(3), 292-296.
- Mu, Y., & Han, S. (2010). Neural oscillations involved in self-referential processing. *Neuroimage*, 53(2), 757-768.
- Nauta, W. J. (1993). The Problem of the Frontal Lobe: A Reinterpretation. In *Neuroanatomy* (pp. 540-560): Birkhauser Boston
- Neville, H. J., & Bavelier, D. (1998). Neural organization and plasticity of language. *Current Opinion in Neurobiology*, 8(2), 254-258.
- Nieuwenhuis, S., Aston-Jones, G., & Cohen, J. D. (2005). Decision making, the P3, and the locus coeruleus--norepinephrine system. *Psychological Bulletin*, 131(4), 510.
- Nolan, H., Whelan, R., & Reilly, R. (2010). "FASTER": Fully Automated Statistical Thresholding for EEG artifact Rejection. *Journal of Neuroscience Methods*, 192(1), 152-162.
- Nolan, H., Whelan, R., & Reilly, R. B. (2010). FASTER: Fully Automated Statistical Thresholding for EEG artifact Rejection. *Journal of Neuroscience Methods*, 192(1), 152-162. doi: 10.1016/j.jneumeth.2010.07.015S0165-0270(10)00389-4 [pii]
- Northoff, G., Wiebking, C., Feinberg, T., & Panksepp, J. (2011). The resting-state hypothesis of major depressive disorder; A translational subcortical-cortical framework for a system disorder. *Neuroscience and Biobehavioral Reviews*, 35(9), 1929-1945.
- Ochsner, K. N., Beer, J. S., Robertson, E. R., Cooper, J. C., Gabrieli, J. D., Kihlstrom, J. F., & D'Esposito, M. (2005). The neural correlates of direct and reflected self-knowledge. *Neuroimage*, 28(4), 797-814.
- Organization, W. H. (2008). *World health statistics 2008*: World Health Organization.
- Osgood, C. E. (1952). The nature and measurement of meaning. *Psychological Bulletin*, 49(3), 197.
- Pandya, D. N., & Barnes, C. L. (1987). *Architecture and connections of the frontal lobe*. Springer US.
- Panksepp, J., & Biven, L. (2012). *The Archaeology of Mind: Neuroevolutionary Origins of Human Emotions (Norton Series on Interpersonal Neurobiology)*: WW Norton & Company.

- Pauly, K., Finkelmeyer, A., Schneider, F., & Habel, U. (2013). The neural correlates of positive self-evaluation and self-related memory. *Social Cognitive and Affective Neuroscience*, 8(8), 878-886.
- Pfeifer, J. H., Lieberman, M. D., & Dapretto, M. (2007). "I know you are but what am I?" The neural bases of self-and social knowledge retrieval in children and adults. *Journal of Cognitive Neuroscience*, 19(8), 1323-1337.
- Phan, K. L., Wager, T., Taylor, S. F., & Liberzon, I. (2002). Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage*, 16(2), 331-348.
- Polich, J. (1998). P300 clinical utility and control of variability. *Journal of Clinical Neurophysiology*, 15(1), 14-33.
- Polich, J. (2007). An integrative theory of P3a and P3b. *Clinical Neurophysiology*, 118, 2128-2148.
- Polich, J. (2007). Updating P300: an integrative theory of P3a and P3b. *Clinical Neurophysiology*, 118(10), 2128-2148. doi: 10.1016/j.clinph.2007.04.019
- Poulsen, C., Luu, P., Crane, S. M., Quiring, J., & Tucker, D. M. (2009). Frontolimbic activity and cognitive bias in major depression. *Journal of Abnormal Psychology*, 118(3), 494-506. doi: 10.1037/a0015920
- Qin, P., & Northoff, G. (2011). How is our self related to midline regions and the default-mode network? *Neuroimage*, 57(3), 1221-1233.
- Rakic, P. (2009). Evolution of the neocortex: a perspective from developmental biology. *Nature Reviews Neuroscience*, 10(10), 724-735.
- Ressler, K. J., & Nemeroff, C. B. (2000). Role of serotonergic and noradrenergic systems in the pathophysiology of depression and anxiety disorders. *Depression and Anxiety*, 12(S1), 2-19.
- Rimes, K. A., & Watkins, E. (2005). The effects of self-focused rumination on global negative self-judgements in depression. *Behaviour Research and Therapy*, 43(12), 1673-1681.
- Robinson, R. G., Kubos, K. I., Starr, L. B., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients: importance of location of lesion. *Brain*, 107(1), 81-93.
- Ross, E. D., & Mesulam, M.-M. (1979). Dominant language functions of the right hemisphere? Prosody and emotional gesturing. *Archives of Neurology*, 36(3), 144.
- Russell, J. A. (1979). Affective space is bipolar. *Journal of Personality and Social Psychology*, 37(3), 345.

- Russell, J. A. (2009). Emotion, core affect, and psychological construction. *Cognition and Emotion*, 23(7), 1259-1283.
- Sackeim, H. A., Luber, B., Katzman, G. P., Moeller, J. R., Prudic, J., Devanand, D., & Nobler, M. S. (1996). The effects of electroconvulsive therapy on quantitative electroencephalograms: relationship to clinical outcome. *Archives of General Psychiatry*, 53(9), 814.
- Sanides, F. (1970). Functional architecture of motor and sensory cortices in primates in the light of a new concept of neocortex evolution. *The Primate Brain: Advances in Primatology*, 1, 137-208.
- Sarsam, M., Parkes, L. M., Roberts, N., Reid, G. S., & Kinderman, P. (2013). The Queen and I: Neural Correlates of Altered Self-Related Cognitions in Major Depressive Episode. *PloS one*, 8(10), e78844.
- Saucier, G., Thalmayer, A. G., Payne, D. L., Carlson, R., Sanogo, L., Ole, ÄKotikash, L., . . . Szarota, P. (2014). A basic bivariate structure of personality attributes evident across nine languages. *Journal of Personality*, 82(1), 1-14.
- Scherg, M. (1990). Fundamentals of dipole source potential analysis. *Advances in Audiology*, 6, 40-69.
- Schwartz, G. E., Davidson, R. J., & Maer, F. (1975). Right hemisphere lateralization for emotion in the human brain: Interactions with cognition. *Science*, 190(4211), 286-288.
- Scott, G. G., O'Donnell, P. J., Leuthold, H., & Sereno, S. C. (2009). Early emotion word processing: Evidence from event-related potentials. *Biological Psychology*, 80(1), 95-104.
- Seeger, C. A., Stone, M., & Keenan, J. P. (2004). Cortical activations during judgments about the self and an other person. *Neuropsychologia*, 42(9), 1168-1177.
- Siegle, G. J., Thompson, W. K., Collier, A., Berman, S. R., Feldmiller, J., Thase, M. E., & Friedman, E. S. (2012). Toward clinically useful neuroimaging in depression treatment: prognostic utility of subgenual cingulate activity for determining depression outcome in cognitive therapy across studies, scanners, and patient characteristics. *Archives of General Psychiatry*, 69(9), 913-924.
- Starkstein, S. E., Fedoroff, P., Berthier, M. L., & Robinson, R. G. (1991). Manic-depressive and pure manic states after brain lesions. *Biological Psychiatry*, 29(2), 149-158.
- Stewart, J. L., Bismark, A. W., Towers, D. N., Coan, J. A., & Allen, J. J. (2010). Resting frontal EEG asymmetry as an endophenotype for depression risk: sex-specific patterns of frontal brain asymmetry. *Journal of Abnormal Psychology*, 119(3), 502.

- Stewart, J. L., Coan, J. A., Towers, D. N., & Allen, J. J. (2011). Frontal EEG asymmetry during emotional challenge differentiates individuals with and without lifetime major depressive disorder. *Journal of Affective Disorders*, 129(1), 167-174.
- Stewart, J. L., Levin-Silton, R., Sass, S. M., Heller, W., & Miller, G. A. (2008). Anger style, psychopathology, and regional brain activity. *Emotion*, 8(5), 701.
- Striedter, G. F. (2005). *Principles of brain evolution*: Sinauer Associates.
- Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science*, 8(3), 204-210.
- Tellegen, A. (1988). The analysis of consistency in personality assessment. *Journal of Personality*, 56(3), 621-663.
- Tellegen, A., Watson, D., & Clark, L. A. (1999). On the dimensional and hierarchical structure of affect. *Psychological Science*, 10(4), 297-303.
- Teneback, C. C., Nahas, Z., Speer, A. M., Molloy, M., Stallings, L. E., Spicer, K. M., . . . George, M. S. (1999). Changes in prefrontal cortex and paralimbic activity in depression following two weeks of daily left prefrontal TMS. *Journal of Neuropsychiatry and Clinical Neurosciences*, 11(4), 426-435.
- Thayer, R. E. (1978). Toward a psychological theory of multidimensional activation (arousal). *Motivation and Emotion*, 2(1), 1-34.
- Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: a meta-analytic review. *Journal of Abnormal Psychology*, 115(4), 715.
- Thompson, E. R. (2007). Development and validation of an internationally reliable short-form of the positive and negative affect schedule (PANAS). *Journal of Cross-Cultural Psychology*, 38(2), 227-242.
- Tomkins, S. S. (1962). Affect, imagery, consciousness: Vol. I. The positive affects.
- Tucker, D. M. (2007). *Mind from Body: Experience from Neural Structure*. New York, NY: Oxford University Press.
- Tucker, D. M., Hartry-Speiser, A., McDougal, L., Luu, P., & Degrandpre, D. (1999). Mood and spatial memory: emotion and right hemisphere contribution to spatial cognition. *Biological Psychology*, 50(2), 103-125.
- Tucker, D. M., Liotti, M., Potts, G. F., Russell, G. S., & Posner, M. I. (1994). Spatiotemporal analysis of brain electrical fields. *Human Brain Mapping*, 1(2), 134-152.

- Tucker, D. M., & Luu, P. (2007). Neurophysiology of motivated learning: adaptive mechanisms underlying cognitive bias in depression. *Cognitive Therapy Research, 31*, 189-209.
- Tucker, D. M., & Luu, P. (2012). *Cognition and Neural Development*. New York, NY: Oxford University Press.
- Tucker, D. M., Luu, P., Desmond, R. E., Harty-Speiser, A., Davey, C., & Flaisch, T. (2003). Corticolimbic mechanisms in emotional decisions. *Emotion, 3*(2), 127-149. doi: 10.1037/1528-3542.3.2.127
- Tucker, D. M., Luu, P., Frishkoff, G., Quiring, J., & Poulsen, C. (2003). Frontolimbic response to negative feedback in depression. *Journal of Abnormal Psychology, 112*, 667-678.
- Tucker, D. M., & Williamson, P. A. (1984). Asymmetric neural control systems in human self-regulation. *Psychology Review, 91*(2), 185-215.
- Walhovd, K. B., Rosquist, H., & Fjell, A. M. (2008). P300 amplitude age reductions are not caused by latency jitter. *Psychophysiology, 45*(4), 545-553.
- Walla, P., Duregger, C., Greiner, K., Thurner, S., & Ehrenberger, K. (2008). Multiple aspects related to self-awareness and the awareness of others: an electroencephalography study. *Journal of Neural Transmission, 115*(7), 983-992.
- Waters, A. C., Song, J. E., Luu, P., & Tucker, D. M. (2012). The Importance of Single Trials: Temporal and Spatial Resolution in Event-Related Potential Research. *Developmental Neuropsychology, 37*(6), 545-558.
- Waters, A. C., & Tucker, D. M. (2012). Neurophysiology of Motivated Learning *Encyclopedia of the Sciences of Learning* (pp. 2455-2457): Springer.
- Waters, A. C., & Tucker, D. M. (2013a). Neurodevelopmental processes of attachment and autonomy. In B. W. Sokol, F. M. E. Grouzet & U. Muller (Eds.), *Self-regulation and autonomy; Social and developmental dimensions of human conduct* (40 ed.): Cambridge University Press.
- Waters, A. C., & Tucker, D. M. (2013b). Positive and negative affect in adolescent self-evaluation: Psychometric information in single trials used to generate dimension-specific ERPs and neural source models. *Psychophysiology, 50*(6), 538-549.
- Watson, Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: the PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063.
- Watson, & Tellegen, A. (1985). Toward a consensual structure of mood. *Psychological Bulletin, 98*(2), 219.

- Watson, D., & Clark, L. A. (1999). The PANAS-X: Manual for the positive and negative affect schedule-expanded form. Retrieved from http://ir.uiowa.edu/psychology_pubs/11
- Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*(1), 3.
- Watson, L. A., Dritschel, B., Obonsawin, M., & Jentsch, I. (2007). Seeing yourself in a positive light: brain correlates of the self-positivity bias. *Brain Research, 1152*, 106-110.
- Weintraub, S., & Mesulam, M. (1987). Right cerebral dominance in spatial attention: Further evidence based on ipsilateral neglect. *Archives of Neurology, 44*(6), 621.
- Whalen, P. J., Rauch, S. L., Etcoff, N. L., McInerney, S. C., Lee, M. B., & Jenike, M. A. (1998). Masked presentations of emotional facial expressions modulate amygdala activity without explicit knowledge. *The Journal of Neuroscience, 18*(1), 411-418.
- Williams, J. B. (1988). A structured interview guide for the Hamilton Depression Rating Scale. *Archives of General Psychiatry, 45*(8), 742-747.
- Yoshimura, S., Okamoto, Y., Onoda, K., Matsunaga, M., Okada, G., Kunisato, Y., . . . Yamawaki, S. (2013). Cognitive behavioral therapy for depression changes medial prefrontal and ventral anterior cingulate cortex activity associated with self-referential processing. *Social Cognitive and Affective Neuroscience*, doi:10.1093/scan/nst009
- Zachar, P., & Ellis, R. D. (2012). Categorical versus Dimensional Models of Affect : A seminar on the theories of Panksepp and Russell. Retrieved from <http://orbis.ebilib.com/patron/FullRecord.aspx?p=939565>
- Zhang, H., Guan, L., Qi, M., & Yang, J. (2013). Self-Esteem Modulates the Time Course of Self-Positivity Bias in Explicit Self-Evaluation. *PloS one, 8*(12), e81169.
- Zhou, A., Li, S., Herbert, C., Xia, R., Xu, K., Xu, Q., . . . Ren, D. (2013). Perspective taking modulates positivity bias in self-appraisals: Behavioral and event-related potential evidence. *Social Neuroscience, 8*(4), 326-333.